```
=> d que stat l29
L15        4045 SEA FILE=HCAPLUS ABB=ON  (?RESPIRATORY?(W)?SYNCYTIAL?(W)?VIRUS?
                OR RSV)
L16        1148 SEA FILE=HCAPLUS ABB=ON  L15 AND (?RECOMB? OR ?VARIANT? OR
                ?MUTANT?)
L17          11 SEA FILE=HCAPLUS ABB=ON  L16 AND (?DELET?(5A)(?SMALL?(W)?HYDROP
                HOB? OR SH))
L18          16 SEA FILE=HCAPLUS ABB=ON  L16 AND ?POLYNUCLEOTID?
L19          26 SEA FILE=HCAPLUS ABB=ON  L17 OR L18
L20          19 SEA FILE=HCAPLUS ABB=ON  L19 AND ?HUMAN?
L21           7 SEA FILE=HCAPLUS ABB=ON  L19 AND ?PARAINFLUENZ?
L22          26 SEA FILE=HCAPLUS ABB=ON  L19 OR L20 OR L21
L25           1 SEA FILE=HCAPLUS ABB=ON  L16 AND ?POLYNUCLEOTID?(5A)(?RESPIRATO
                RY?(W)?SYNCYTIAL?(W)?VIRUS? OR RSV)
L29          26 SEA FILE=HCAPLUS ABB=ON  L22 OR L25

=> d ibib abs l29 1-26

L29  ANSWER 1 OF 26  HCAPLUS  COPYRIGHT 2003 ACS
ACCESSION NUMBER:        2003:291120  HCAPLUS
TITLE:                   Respiratory syncytial
                         virus (RSV) fusion protein subunit
                         F2, not attachment protein G, determines the
                         specificity of RSV infection
AUTHOR(S):               Schlender, Jorg; Zimmer, Gert; Herrler, Georg;
                         Conzelmann, Karl-Klaus
CORPORATE SOURCE:        Max von Pettenkofer Institute and Gene Center,
                         Ludwig-Maximilians-University Munich, Munich, D-81377,
                         Germany
SOURCE:                  Journal of Virology (2003), 77(8), 4609-4616
                         CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER:               American Society for Microbiology
DOCUMENT TYPE:           Journal
LANGUAGE:                English
AB   Human respiratory syncytial virus
     (HRSV) and bovine RSV (BRSV) infect human beings and
     cattle in a species-specific manner. We have here analyzed the
     contribution of RSV envelope proteins to species-specific entry
     into cells. In contrast to permanent cell lines, primary cells of
     human or bovine origin, including differentiated respiratory
     epithelia, peripheral blood lymphocytes, and macrophages, showed a
     pronounced species-specific permissivity for HRSV and BRSV infection,
     resp. Recombinant BRSV deletion mutants
     lacking either the small hydrophobic (SH) protein gene
     or both SH and the attachment glycoprotein (G) gene retained their
     specificity for bovine cells, whereas corresponding mutants
     carrying the HRSV F gene specifically infected human cells. To
     further narrow the responsible region of F, two reciprocal chimeric F
     constructs were assembled from BRSV and HRSV F1 and F2 subunits. The
     specificity of recombinant RSV carrying only the
     chimeric F proteins strictly correlated with the origin of the
     membrane-distal F2 domain. A contribution of G to the specificity of
     entry could be excluded after reintroduction of BRSV or HRSV G. Virus
     with F1 and G from BRSV and with only F2 from HRSV specifically infected
     human cells, whereas virus expressing F1 and G from HRSV and F2
     from BRSV specifically infected bovine cells. The introduction of G
     enhanced the infectivities of both chimeric viruses to equal degrees.
     Thus, the role of the nominal attachment protein G is confined to
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facilitating infection in a non-species-specific manner, most probably by binding to cell surface glycosaminoglycans. The identification of the F2 subunit as the determinant of **RSV** host cell specificity facilitates identification of virus receptors and should allow for development of reagents specifically interfering with **RSV** entry.

REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 2 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2003:282705 HCAPLUS
DOCUMENT NUMBER: 138:282319
TITLE: Construction of **recombinant respiratory syncytial viruses** with deleted surface glycoprotein genes and uses as vaccine
INVENTOR(S): Wertz, Gail W.; Megaw, George; Oomens, Tom A.
PATENT ASSIGNEE(S): UAB Research Foundation, USA
SOURCE: PCT Int. Appl., 80 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003029416	A2	20030410	WO 2002-US31086	20021001
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2003072773	A1	20030417	US 2002-262238	20021001

PRIORITY APPLN. INFO.: US 2001-326259P P 20011001
US 2002-397289P P 20020719

AB The present invention provides **recombinant respiratory syncytial viruses** (**RSV**) in which all of the surface glycoprotein genes encoding the attachment protein G, the fusion protein F, and the **Small Hydrophobic** protein **SH** are **deleted**. The genes are replaced by a chimeric gene encoding a heterologous entry protein derived from the Vesicular Stomatitis Virus G protein or GP64 of baculovirus. Alternatively, the replacement proteins are provided in trans. Marker genes such as those encoding .beta.-glucuronidase (GUS) and green fluorescent protein (EGFP) are also added to the upstream and downstream, side of hybrid gene for easy detection. These infectious **recombinant respiratory syncytial viruses** offer alternatives and improvements as vaccine candidates.

L29 ANSWER 3 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:906448 HCAPLUS
DOCUMENT NUMBER: 138:12025
TITLE: Construction of retroviral vectors, their infection of neuron cells and therapeutical uses thereof
INVENTOR(S): Edelman, Gerald M.; Owens, Geoffrey

PATENT ASSIGNEE(S): The Scripps Research Institute, USA
SOURCE: PCT Int. Appl., 122 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002094989	A2	20021128	WO 2002-US15816	20020517

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-292201P P 20010518
US 2001-334972P P 20011130

AB A retroviral vector plasmid that contains a retrovirus long terminal repeat, including a U5 region, an R region and a U3 region, which contains a transcriptional regulatory element or a site for inserting such an element; a constitutive transport element, which is heterologous to the U5 region and lacks any translation start codons; and a cloning site, and that substantially lacks any translation start codons between the R region and the cloning site, is provided. In addn., retroviral vector based on the retroviral vector plasmid, and a retroviral vector genome is provided, as is a cell contg. the retroviral vector plasmid or retroviral vector. Also provided is a kit contg. such a retroviral vector plasmid or retroviral vector. Methods of making and methods of using such a retroviral vector plasmid or retroviral vector also are provided, including, for example, methods of using the vector or vector plasmid to introduce an expressible **polynucleotide** into a cell such as a neural stem cell.

L29 ANSWER 4 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:778642 HCAPLUS
DOCUMENT NUMBER: 137:293542
TITLE: Microparticles and methods for delivery of **recombinant** viral vaccines
INVENTOR(S): Hural, John; Johnson, Mark E.; Spies, A. Gregory
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 15 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002146828	A1	20021010	US 2002-40990	20020107
WO 2002092132	A2	20021121	WO 2002-US235	20020107

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,

LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ,
UA, UG, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH,
CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR,
BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2001-260164P P 20010105
US 2001-333701P P 20011127

AB Disclosed is a viral vector conjugated to a microparticle, wherein the viral vector comprises a **polynucleotide** encoding a heterologous polypeptide. Conjugation of the viral vector to the microparticle results in a dramatic increase in the efficacy of the elicited immune response. The microparticle has a characteristic length of about 0.5 .mu.m to about 20 .mu.m, comprising a cationic lipid, a polymer of a natural or synthetic monomer, or an anionic surfactant. Also disclosed is a method for delivering a **polynucleotide** to a cell comprising contacting the cell with a viral vector of the invention. In a preferred embodiment, the cell is an antigen-presenting cell, such as a dendritic cell. The invention further provides a vaccine comprising a viral vector of the invention. The methods is demonstrated by delivering Mycobacterium tuberculosis single antigen or multiple antigens to APC or dendritic cell. The invention thus provides a method for delivering a **polynucleotide** to a subject, a method of stimulating an immune response in a subject, a method of treating cancer in a subject, a method of inhibiting tumor growth in a subject, and a method of treating an infection in a subject.

L29 ANSWER 5 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:429041 HCAPLUS
DOCUMENT NUMBER: 137:19372
TITLE: **Recombinant respiratory syncytial virus** comprising deleted or attenuated accessory gene(s) for use as vaccines and vaccine expression systems
INVENTOR(S): Jin, Hong; Tang, Roderick; Li, Shengqiang; Bryant, Martin
PATENT ASSIGNEE(S): Aviron, Inc., USA
SOURCE: PCT Int. Appl., 150 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002044334	A2	20020606	WO 2001-US44819	20011128
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2002036522	A5	20020611	AU 2002-36522	20011128

PRIORITY APPLN. INFO.: US 2000-724416 A 20001128
WO 2001-US44819 W 20011128

AB The present invention relates to genetically engineered

recombinant respiratory syncytial virus and viral vectors which contain deletions of various viral accessory gene(s) either singly or in combination. In accordance with the present invention, the **recombinant** respiratory syncytial viral vectors and viruses are engineered to contain complete deletions of the M2-2, NS1, NS2, or SH viral accessory genes or various combinations thereof. In addn., the present invention relates to the attenuation of **respiratory syncytial virus** by mutagenesis of the M2-1 gene.

L29 ANSWER 6 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:10511 HCAPLUS
DOCUMENT NUMBER: 136:84681
TITLE: **Respiratory syncytial virus** vaccines expressing protective antigens from promotor-proximal genes
INVENTOR(S): Krempl, Christine D.; Collins, Peter L.; Murphy, Brian R.; Buchholz, Ursula; Whitehead, Stephen S.
PATENT ASSIGNEE(S): The Government of the United States of America, USA
SOURCE: PCT Int. Appl., 168 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002000693	A2	20020103	WO 2001-US20107	20010622
WO 2002000693	A3	20020926		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 2002146433	A1	20021010	US 2001-887469	20010622
EP 1294858	A2	20030326	EP 2001-946696	20010622
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				

PRIORITY APPLN. INFO.: US 2000-213708P P 20000623
WO 2001-US20107 W 20010622

AB **Recombinant respiratory syncytial virus** (**RSV**) having the position of genes shifted within the genome or antigenome of the **recombinant** virus are infectious and attenuated in **humans** and other mammals. Gene shifted **RSV** are constructed by insertion, deletion or rearrangement of genes or genome segments within the **recombinant** genome or antigenome and are useful in vaccine formulations for eliciting an anti-**RSV** immune response. Also provided are isolated **polynucleotide** mols. and vectors incorporating a **recombinant RSV** genome or antigenome wherein a gene or gene segment is shifted to a more promoter-proximal or promoter-distal position within the genome or antigenome compared to a wild type position of the gene in the **RSV** gene map. Shifting the position of genes in this manner provides for a selected increase or decrease in expression of the gene, depending on the nature and degree of the positional shift.

In one embodiment, **RSV** glycoproteins are upregulated by shifting one or more glycoprotein-encoding genes to a more promoter-proximal position. Genes of interest for manipulation to create gene position-shifted **RSV** include any of the NS1, NS2, N, P, M, SH, M2(ORF1), M2(ORF2), L, F or G genes or a genome segment that may be part of a gene or extragenic. A variety of addnl. mutations and nucleotide modifications are provided within the gene position-shifted **RSV** of the invention to yield desired phenotypic and structural effects.

L29 ANSWER 7 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:544284 HCAPLUS
DOCUMENT NUMBER: 135:254313
TITLE: Functional analysis of **recombinant respiratory syncytial virus deletion mutants** lacking the **small hydrophobic** and/or attachment glycoprotein gene
AUTHOR(S): Techaarpornkul, Sunee; Barretto, Naina; Peeples, Mark E.
CORPORATE SOURCE: Department of Immunology/Microbiology, Rush-Presbyterian-St. Luke's Medical Center, Chicago, IL, 60612, USA
SOURCE: Journal of Virology (2001), 75(15), 6825-6834 CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Respiratory syncytial virus** (**RSV**) produces 3 envelope glycoproteins, the attachment glycoprotein (G), the fusion (F) protein, and the small hydrophobic (SH) protein. It had been assumed, by analogy with other paramyxoviruses, that the G and F proteins would be required for the 1st 2 steps of viral entry, attachment and fusion. However, following repeated passage in cell culture, a viable **mutant RSV** that lacked both the G and SH genes was isolated. To explore the roles of the G, F, and SH proteins in virion assembly, function, and cytopathol., the full-length **RSV** cDNA was modified and used to rescue infectious **RSV** lacking the G and/or SH genes. The 3 resulting viruses and the parental virus all contain the green fluorescent protein (GFP) gene that serves to identify infected cells. Purified, radiolabeled virions were used to examine virus prodn. and function, in conjunction with GFP to quantify infected cells. The G protein enhanced virion binding to target cells but played no role in penetration after attachment. The G protein also enhanced cell-to-cell fusion, presumably via cell-to-cell binding, and enhanced virion assembly or release. The presence or absence of the G protein in virions has no obvious effect on the content of F protein or host cell proteins in the virion. In growth curve expts., the viruses lacking the G protein produced viral titers that were at least 10-fold lower than titers of viruses contg. the G protein. This redn. is due in large part to the less efficient release of virions and the lower infectivity of the released virions. In the absence of the G protein, virus expressing both the F and SH proteins displayed somewhat smaller plaques, lower fusion activity, and slower viral entry than the virus expressing the F protein alone, suggesting that the SH protein has a neg. effect on virus fusion in cell culture.
REFERENCE COUNT: 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 8 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:537386 HCAPLUS

DOCUMENT NUMBER: 135:134612
TITLE: Formation of infectious **respiratory syncytial virus** particles by expression of cloned viral genes
INVENTOR(S): Collins, Peter L.
PATENT ASSIGNEE(S): United States Dept. of Health and Human Services, USA
SOURCE: U.S., 24 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6264957	B1	20010724	US 1996-720132	19960927
US 2002182228	A1	20021205	US 2001-847173	20010503

PRIORITY APPLN. INFO.: US 1995-7083P P 19950927
US 1996-720132 A3 19960927

AB Isolated **polynucleotide** mols. provide a DNA and RNA genomes and antigenomes of **respiratory syncytial viruses** (**RSV**), including those of **human**, bovine or murine **RSV** or **RSV**-like viruses, and chimera thereof that can be used to manuf. infectious particles of the virus. The **recombinant** genome or antigenome can be expressed with a nucleocapsid (N) protein, a nucleocapsid phosphoprotein (P), a large (L) polymerase protein, and an RNA polymerase elongation factor to produce isolated infectious **RSV** particles. The genome may be on a single vector or as a group of expression units on a no. of vectors. The **recombinant** **RSV** genome and antigenome can be modified to produce desired phenotypic changes, such as attenuated viruses for vaccine use. A cDNA encoding a genome of **RSV** A2 was constructed and a T7 RNA polymerase promoter placed upstream of the genome. This was introduced into an Hep2 cells line expressing the bacteriophage T7 RNA polymerase gene. After three days a culture supernatant was used to infect Hep-2 cells and five days after infection plaques showing the morphol. similar to **RSV** plaques and abundant F protein were obtained. Plaque-purified virus was analyzed by RT-PCR and showed the expected pattern of amplification products. **Variants** of the virus with low plating efficiency or carrying a reporter gene were propagated by the same means.

REFERENCE COUNT: 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 9 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:435250 HCAPLUS
DOCUMENT NUMBER: 135:41798
TITLE: **Recombinant parainfluenza** viruses as vectors to protect against infection and disease caused by **parainfluenza** viruses and other **human** pathogens
INVENTOR(S): Murphy, Brian R.; Collins, Peter L.; Schmidt, Alexander C.; Durbin, Anna P.; Skiadopoulos, Mario H.; Tao, Tao
PATENT ASSIGNEE(S): The Government of the United States of America, As Represented by the Department of Health and Human Services, USA
SOURCE: PCT Int. Appl., 305 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001042445	A2	20010614	WO 2000-US33293	20001208
WO 2001042445	A3	20011129		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 2001020731	A5	20010618	AU 2001-20731	20001208
EP 1179054	A2	20020213	EP 2000-984052	20001208
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, MC, IE, SI, LT, LV, FI, RO				

PRIORITY APPLN. INFO.:
US 1999-170195P P 19991210
US 1999-458813 A 19991210
US 1999-459062 A 19991210
WO 2000-US33293 W 20001208

AB Chimeric **parainfluenza** viruses (PIVs) are provided that incorporate a PIV vector genome or antigenome and one or more antigenic determinant(s) of a heterologous PIV or non-PIV pathogen. These chimeric viruses are infectious and attenuated in **humans** and other mammals and are useful in vaccine formulations for eliciting an immune responses against one or more PIVs, or against a PIV and non-PIV pathogen. Also provided are isolated **polynucleotide** mols. and vectors incorporating a chimeric PIV genome or antigenome which includes a partial or complete PIV vector genome or antigenome combined or integrated with one or more heterologous gene(s) or genome segment(s) encoding antigenic determinant(s) of a heterologous PIV or non-PIV pathogen. In preferred aspects of the invention, chimeric PIV incorporate a partial or complete **human**, bovine, or **human**-bovine chimeric, PIV vector genome or antigenome combined with one or more heterologous gene(s) or genome segment(s) from a heterologous PIV or non-PIV pathogen, wherein the chimeric virus is attenuated for use as a vaccine agent by any of a variety of mutations and nucleotide modifications introduced into the chimeric genome or antigenome. Chimeric **recombinant** PIV3s bearing an antigenic determinant of measles virus replicate efficiently in hamsters and induce high titers of antibodies against both HPIV3 and measles.

L29 ANSWER 10 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:338740 HCAPLUS
DOCUMENT NUMBER: 134:348962
TITLE: Transfection system and expression vectors used for **recombinant** production of heterologous proteins (such as CAB-2, CAB-4, uPAR, VEGF-D or viral glycoprotein) in mammalian and/or insect cells
INVENTOR(S): Innis, Michael; Scott, Elizabeth
PATENT ASSIGNEE(S): Chiron Corporation, USA
SOURCE: PCT Int. Appl., 71 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001032901	A1	20010510	WO 1999-US31275	19991230
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6316253	B1	20011113	US 1999-475460	19991230
EP 1228237	A1	20020807	EP 1999-967788	19991230
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
JP 2003513635	T2	20030415	JP 2001-535583	19991230
US 2001024807	A1	20010927	US 2000-748061	20001222
US 6451539	B2	20020917		
US 2003064517	A1	20030403	US 2002-146356	20020513

PRIORITY APPLN. INFO.:

US 1999-162930P	P	19991101
US 1999-475460	A	19991230
US 1999-162980P	P	19991101
WO 1999-US31275	W	19991230
US 2000-748061	A1	20001222

AB The invention provides a transfection system for **recombinant** prodn. of heterologous proteins (such as CAB-2, CAB-4, uPAR, VEGF-D or a viral glycoprotein) in mammalian and/or insect cells. The invention relates that the transfection system comprises two DNA constructs (plasmids), which are used to transform said mammalian and/or insect cells. The first DNA construct (plasmid) contains a selectable marker, such as a functionally impaired neomycin phosphotransferase II gene (neo*), and a second marker, such as a dihydrofolate reductase gene (dhfr) which contains at least one disabling mutation in the 5' coding region. The second DNA construct (plasmid) contains the **polynucleotide** sequence encoding the heterologous protein, and a third selectable marker, such as a dhfr gene, which contains a mutation at the 3' coding region. The invention also relates that the DNA constructs (plasmids) contain Cytomegalovirus, Rous sarcoma virus and Simian virus 40 promoters, polyadenylation sites and origin of replications. The invention further relates that in cells transformed with said DNA constructs (plasmids) **recombination** occurs between the second and third selectable markers (the mutated dhfr genes), which results in cells being able to express the heterologous protein. The invention also provides the expression vectors used to transform said mammalian and/or insect cells. As way of illustration, the materials and methods claimed in this invention were used to **recombinantly** produce CAB2.1 and CAB4.2 in CHO cells.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 11 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:211559 HCAPLUS
DOCUMENT NUMBER: 135:299395
TITLE: **Respiratory syncytial virus** can tolerate an intergenic sequence of at least 160 nucleotides with little effect on

transcription or replication in vitro and in vivo
AUTHOR(S): Bukreyev, Alexander; Murphy, Brian R.; Collins, Peter L.
CORPORATE SOURCE: Laboratory of Infectious Diseases, National Institute of Allergy and Infectious Diseases, Bethesda, MD, 20892-0720, USA
SOURCE: Journal of Virology (2000), 74(23), 11017-11026
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The intergenic sequences (IGS) between the first nine genes of **human respiratory syncytial virus** (**RSV**) vary in length from 1 to 56 nucleotides and lack apparent conserved sequence motifs. To investigate their influence on sequential transcription and viral growth, **recombinant RSV** strain A2, from which the **SH** gene had been **deleted** to facilitate manipulation, was further modified to contain an M-G IGS of 16, 30, 44, 58, 65, 72, 86, 100, 120, 140, or 160 nucleotides. All of the viruses were viable. For viruses with an M-G IGS of 100 nucleotides or more, plaque size decreased with increasing IGS length. In this same length range, increasing IGS length was assocd. with modest attenuation during single-step, but not multistep, growth in HEp-2 cells. Surprisingly, Northern blot anal. of the accumulation of six different mRNAs indicated that there was little or no change in transcription with increasing IGS length. Thus, the **RSV** polymerase apparently can readily cross IGS of various lengths, including unnaturally long ones, with little or no effect on the efficiency of termination and reinitiation. This finding supports the view that the IGS do not have much effect on sequential transcription and provides evidence from infectious virus that IGS length is not an important regulatory feature. To evaluate replication in vivo, BALB/c mice were infected intranasally with **RSV** contg. an M-G IGS of 65, 140, or 160 nucleotides. Replication of the latter two viruses was decreased up to 5- and 25-fold in the upper and lower respiratory tracts, resp., on day 3 following infection. However, the level of replication at both sites on days 4 and 5 was very similar to that of the virus with an IGS of 65 nucleotides. Thus, the modest attenuation in vivo assocd. with the longer IGS was additive to that conferred by **deletion** of the **SH** gene and might be useful to incrementally increase the level of attenuation of a live-attenuated vaccine virus.
REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 12 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:160042 HCAPLUS
DOCUMENT NUMBER: 134:323301
TITLE: **Recombinant** bovine **respiratory syncytial virus** with **deletions** of the G or **SH** genes: G and F proteins bind heparin
AUTHOR(S): Karger, Axel; Schmidt, Ulrike; Buchholz, Ursula J.
CORPORATE SOURCE: Institute of Molecular Biology, Federal Research Centre for Virus Diseases of Animals, Friedrich-Loeffler-Institutes, Insel Riems, D-17498, Germany
SOURCE: Journal of General Virology (2001), 82(3), 631-640
CODEN: JGVIAY; ISSN: 0022-1317
PUBLISHER: Society for General Microbiology
DOCUMENT TYPE: Journal

LANGUAGE: English

AB Bovine **respiratory syncytial virus** (BRSV) encodes three transmembrane envelope glycoproteins, namely the small hydrophobic (SH) protein, the attachment glycoprotein (G) and the fusion glycoprotein (F). The BRSV reverse genetics system has been used to generate viable **recombinant** BRSV lacking either the G gene or the SH gene or both genes. The deletion **mutants** were fully competent for multicycle growth in cell culture, proving that, of the BRSV glycoprotein genes, the SH and G genes are non-essential. Virus morphogenesis was not impaired by either of the deletions. The deletion **mutants** were used to study the role of the F glycoprotein and the contributions of SH and G with respect to virus attachment. Attachment mediated by the F protein alone could be blocked by sol. heparin, but not by chondroitin sulfate. Heparin affinity chromatog. revealed that both the BRSV G and F glycoproteins have heparin-binding activity, with the affinity of the F glycoprotein being significantly lower than that of G. Therefore, the roles of the BRSV glycoproteins in virus attachment and receptor binding have to be reconsidered.

REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 13 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:101297 HCAPLUS
DOCUMENT NUMBER: 134:142758
TITLE: Rescue of mumps virus from cDNA
INVENTOR(S): Clarke, David K.; Johnson, Erik J.; Sidhu, Mohinderjit S.; Udem, Stephen A.
PATENT ASSIGNEE(S): American Home Products Corporation, USA
SOURCE: PCT Int. Appl., 133 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001009309	A2	20010208	WO 2000-US21192	20000802
WO 2001009309	A3	20010517		
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1218499	A2	20020703	EP 2000-952452	20000802
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				

PRIORITY APPLN. INFO.:
US 1999-146664P P 19990802
US 2000-213654P P 20000623
WO 2000-US21192 W 20000802

AB This invention relates to a method for **recombinantly** producing, via rescue of mumps virus, a nonsegmented, neg.-sense, single-stranded RNA virus, and immunogenic compns. formed therefrom. Addnl. embodiments relate to methods of producing the mumps virus as an attenuated and/or infectious virus. The **recombinant** viruses are prepd. from cDNA clones, and, accordingly, viruses having defined changes, including

nucleotide/**polynucleotide** deletions, insertions, substitutions and rearrangements, in the place of the genome are obtained. Isolated, amplified and sequenced mumps viral RNA was used in construction of expression plasmids and a minireplicon. Expression plasmids (pMUVNP, pMUVP, and pMUVL) for mumps virus NP, P, and L proteins were constructed by splicing cDNA for each gene between T7 RNA polymerase promoter and the T7 RNA polymerase transcription termination sequence of modified plasmid vector pEMC. A synthetic minireplicon MUVCAT was constructed using the T7 RNA polymerase promoter to start transcription with the exact mumps virus 5'-terminal nucleotide and a hepatitis delta virus ribozyme sequence was positioned to generate the precise mumps virus 3'-terminal nucleotide in minireplicon RNA transcripts. In MUVCAT, the bacterial acetyl transferase gene CAT replaces all coding and intercistronic sequences between the 3' mumps virus leader and the untranslated region of gene NP and the 5'-trailer and the untranslated region of gene L. A full-length genome cDNA (pMUVFL) for the mumps virus was assembled by successive cloning of fragments into the same plasmid backbone used to construct the plasmid pMUVCAT. Rescue of CAT activity was demonstrated when cells were transfected with pMUVCAT either with pMUVNP, pMUVP, and pMUVL or with mumps virus. Full-length mumps virus could also be rescued from cells transfected with pMUVFL, pMUVNP, pMUVP, and pMUVL.

L29 ANSWER 14 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:742232 HCAPLUS
DOCUMENT NUMBER: 133:306354
TITLE: Production of attenuated negative stranded RNA virus vaccines from cloned nucleotide sequences
INVENTOR(S): Murphy, Brian R.; Collins, Peter L.; Durbin, Anna P.; Skiadopoulos, Mario H.
PATENT ASSIGNEE(S): United States Dept. of Health and Human Services, USA
SOURCE: PCT Int. Appl., 137 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000061737	A2	20001019	WO 2000-US9695	20000412
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1171623	A2	20020116	EP 2000-922075	20000412
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
BR 2000011159	A	20020723	BR 2000-11159	20000412
JP 2002541798	T2	20021210	JP 2000-611661	20000412

PRIORITY APPLN. INFO.: US 1999-129006P P 19990413
WO 2000-US9695 W 20000412

AB Attenuated, **recombinant** neg. stranded RNA viruses suitable for vaccine use are produced from one or more isolated **polynucleotide** mols. encoding the virus. A **recombinant** genome or antigenome of the subject virus is modified to encode a mutation within a

recombinant protein of the virus at one or more amino acid positions(s) corresponding to a site of an attenuating mutation in a heterologous, **mutant** neg. stranded RNA virus. A similar attenuating mutation as identified in the heterologous neg. stranded RNA virus is thus incorporated at a corresponding site within the **recombinant** virus to confer an attenuated phenotype on the **recombinant** virus. The attenuating mutation incorporated in the **recombinant** virus may be identical or conservative in relation to the attenuating mutation identified in the heterologous, **mutant** virus. By the transfer of mutations into **recombinant** neg. stranded RNA viruses in this manner, candidate vaccine viruses are engineered to elicit a desired immune response against a subject virus in a host susceptible to infection thereby. Attenuating mutations identified in **human parainfluenza** virus type 3 (HPIV3) JS cp45 and **respiratory syncytial virus** cp530 and Sendai virus were mapped to conserved sequence elements among heterologous neg. stranded RNA viruses. Introduction of the F456L mutation in the L polymerase of **recombinant** cp45-based attenuated viruses increases temp. sensitivity. Heterologous transfer of an attenuating mutation in the C protein of Sendai virus into a **recombinant** HPIV3 vaccine candidate was also achieved.

L29 ANSWER 15 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:467544 HCAPLUS
DOCUMENT NUMBER: 133:249519
TITLE: **Recombinant respiratory syncytial viruses** with **deletions** in the NS1, NS2, **SH**, and M2-2 genes are attenuated in vitro and in vivo
AUTHOR(S): Jin, Hong; Zhou, Helen; Cheng, Xing; Tang, Roderick; Munoz, Mary; Nguyen, Nghia
CORPORATE SOURCE: Aviron, Mountain View, CA, 94043, USA
SOURCE: Virology (2000), 273(1), 210-218
CODEN: VIRLAX; ISSN: 0042-6822
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB **Respiratory syncytial virus** (**RSV**) encodes several proteins that lack well-defined functions; these include NS1, NS2, SH, and M2-2. Previous work has demonstrated that NS2, SH, and M2-2 can each be deleted from **RSV** genome and thus are considered as accessory proteins. To det. whether **RSV** can replicate efficiently when two or more transcriptional units are **deleted**, the authors removed NS1, NS2, **SH**, and M2-2 genes individually and in different combinations from an infectious cDNA clone derived from **human RSV** A2 strain. The following six **mutants** with two or more genes deleted were obtained: .DELTA.NS1NS2, .DELTA.M2-2SH, .DELTA.M2-2NS2, .DELTA.SHNS1, .DELTA.SHNS2, and .DELTA.SHNS1NS2. Deletion of M2-2 together with NS1 was detrimental to **RSV** replication. It was not possible to obtain a **recombinant RSV** when all four genes were deleted. All of the double and triple deletion **mutants** exhibited reduced replication and small plaque morphol. in vitro. Replication of these deletion **mutants** was more reduced in HEp-2 cells than in Vero cells. Among the 10 single and multiple gene deletion **mutants** obtained, .DELTA.M2-2NS2 was most attenuated. .DELTA.M2-2NS2 formed barely visible plaques in HEp-2 cells and had a redn. of titer of 3 log10 compared with the wild-type **recombinant RSV** in infected HEp-2 cells. When inoculated intranasally into cotton rats, all of the deletion **mutants** were attenuated in the respiratory

tract. The data indicated that the NS1, NS2, SH, and M2-2 proteins, although dispensable for virus replication in vitro, provide auxiliary functions for efficient **RSV** replication. (c) 2000 Academic Press.

REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 16 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:406926 HCAPLUS
DOCUMENT NUMBER: 133:162910
TITLE: Role of Type I IFNs in the in Vitro Attenuation of Live, Temperature-Sensitive Vaccine Strains of **Human Respiratory Syncytial Virus**
AUTHOR(S): Loveys, Deborah A.; Kulkarni, Sandhya; Atreya, Prabha L.
CORPORATE SOURCE: Laboratory of Pediatric and Respiratory Viral Diseases, Food and Drug Administration, Bethesda, MD, 20892, USA
SOURCE: Virology (2000), 271(2), 390-400
CODEN: VIRLAX; ISSN: 0042-6822
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The contributions of type I interferons (IFNs) to the in vitro attenuation of 3 temp.-sensitive (Ts) subgroup A and 1 subgroup B deletion **mutant RSV** strains were evaluated. The ability of these vaccine viruses to induce IFNs at their permissive and restrictive temps. and their sensitivity to the antiviral effects of exogenous I IFNs were tested in **human** lung epithelial A549 cells. The authors' results show that the highly attenuated and immunogenic subgroup A vaccine strain Ts1C produced higher levels of IFN-.beta. than its parent RSS-2 or 2 related strains, Ts1A and Ts1B, at their permissive temp. Growth of **RSV**-infected A549 cultures at restrictive temps. or prior UV inactivation of the virus abolished the obsd. induction of IFN-.beta., suggesting a strict requirement of viral replication for cellular IFN induction. The enhanced induction of IFN-.beta. by the highly immunogenic Ts1C at permissive temp. may be an advantageous characteristic of a live intranasal vaccine candidate. The subgroup B strain **RSV** B1 and its **mutant** cp-52 (with **SH** and G gene **deletions**) both induced similar but low levels of IFN-.beta.. Hence the obsd. over-attenuation of cp-52 in **human** infants is probably not due to enhanced IFN induction during its replication in the host. The ability of cp-52, which does not express the SH and G proteins, to induce IFN-.beta. levels similar to those of its parent strain suggests that these viral proteins may not have a role in the induction of IFN-.beta. in the host. In addn., both subgroup A and B **mutants** and their resp. parent strains were similarly resistant to the antiviral effects of exogenous IFN-.alpha. or -.beta.. Therefore, increased sensitivity of the **mutants** to IFNs does not seem to contribute to their attenuation. (c) 2000 Academic Press.

REFERENCE COUNT: 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 17 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:227533 HCAPLUS
DOCUMENT NUMBER: 132:264094
TITLE: **Mutant** cholera holotoxin as an adjuvant
INVENTOR(S): Holmes, Randall K.; Jobling, Michael G.; Eldridge, John H.; Green, Bruce A.; Hancock, Gerald E.; Peek,

Joel A.
PATENT ASSIGNEE(S): American Cyanamid Company, USA; Uniformed Services University of the Health Sciences
SOURCE: PCT Int. Appl., 152 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000018434	A1	20000406	WO 1999-US22520	19990930
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2344740	AA	20000406	CA 1999-2344740	19990930
AU 9964039	A1	20000417	AU 1999-64039	19990930
BR 9914160	A	20010626	BR 1999-14160	19990930
EP 1117435	A1	20010725	EP 1999-951639	19990930
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2002525093	T2	20020813	JP 2000-571951	19990930

PRIORITY APPLN. INFO.: US 1998-102430P P 19980930
WO 1999-US22520 W 19990930

AB A **mutant** cholera holotoxin featuring a point mutation at amino acid 29 of the A subunit, wherein the glutamic acid residue is replaced by an amino acid other than aspartic acid, is useful as an adjuvant in an antigenic compn. to enhance the immune response in a vertebrate host to a selected antigen from a pathogenic bacterium, virus, fungus or parasite. In a particular embodiment, the amino acid 29 is histidine. The **mutant** cholera holotoxin may contain at least one addnl. mutation in the A subunit at a position other than amino acid 29. The antigenic compn. may include a second adjuvant in addn. to the **mutant** cholera holotoxin.

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 18 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:784242 HCAPLUS
DOCUMENT NUMBER: 132:20813
TITLE: Novel methods for rescue of RNA viruses
INVENTOR(S): Parks, Christopher L.; Sidhu, Mohinderjit S.; Udem, Stephen A.; Kovacs, Gerald R.
PATENT ASSIGNEE(S): American Cyanamid Company, USA
SOURCE: PCT Int. Appl., 75 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

WO 9963064 A1 19991209 WO 1999-US12292 19990603
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
CA 2333313 AA 19991209 CA 1999-2333313 19990603
AU 9944144 A1 19991220 AU 1999-44144 19990603
BR 9910929 A 20010220 BR 1999-10929 19990603
EP 1090108 A1 20010411 EP 1999-927175 19990603
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, PT, IE, FI
PRIORITY APPLN. INFO.: US 1998-87800P P 19980603
US 1998-87800 P 19980603
WO 1999-US12292 W 19990603

AB This invention relates to improved methods for producing nonsegmented, neg.-sense, single-stranded RNA viruses of the Order designated Mononegavirales virus, including embodiments relating to methods of producing such viruses as attenuated and/or infectious viruses, such as Measles virus (MV) and **respiratory syncytial virus** (**RSV**). One method for producing a **recombinant** virus from the Order Mononegavirales comprises (a) in at least one host cell, conducting transfection of a rescue compn. which comprises (i) a transcription vector comprising an isolated nucleic acid mol. which comprises **polynucleotide** sequence encoding a genome or antigenome of a nonsegmented, neg.-sense, single stranded RNA virus of the Order Mononegavirales and (ii) at least one expression vector which comprises at least one isolated nucleic acid mol. encoding the trans-acting proteins necessary for encapsidation, transcription and replication; in a host cell under conditions sufficient to permit the co-expression of these vectors and the prodn. of the **recombinant** virus; (b) heating the transfected rescue compn. to an effective heat shock temp. under conditions sufficient to increase the recovery of the **recombinant** virus; and optionally, (c) harvesting the resulting **recombinant** virus.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 19 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:764219 HCAPLUS
DOCUMENT NUMBER: 132:9634
TITLE: **Recombinant** AAV vector expressing truncated **human** factor VIII for gene therapy of hemophilia A
INVENTOR(S): Snyder, Richard; Dull, Thomas J.; Mcguinness, Ryan; Finer, Mitchell H.
PATENT ASSIGNEE(S): Cell Genesys, Inc., USA
SOURCE: PCT Int. Appl., 54 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9961642	A1	19991202	WO 1999-US10473	19990527

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 2002155580	A1	20021024	US 1998-84425	19980527
CA 2329505	AA	19991202	CA 1999-2329505	19990527
AU 9941857	A1	19991213	AU 1999-41857	19990527
EP 1082446	A1	20010314	EP 1999-925607	19990527
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2002516114	T2	20020604	JP 2000-551025	19990527

PRIORITY APPLN. INFO.: US 1998-84425 A 19980527
WO 1999-US10473 W 19990527

AB Claimed are methods and materials for expressing a polypeptide with factor VIII activity comprising administering an rAAV vector contg. a plasmid encoding a truncated version of **human** factor VIII, contg., for example, a 90 kD heavy chain of factor VIII fused to a light chain of factor VIII. In a preferred embodiment, the vector comprises a liver-specific promoter such as from **human** factor IX or Rous sarcoma virus, the factor VIII-specifying **polynucleotide**, and a polyadenylation site, with the untranslated region of cytomegalovirus and the .alpha.-globin splice site sepg. the promoter from the coding **polynucleotide**.

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 20 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:708498 HCAPLUS
DOCUMENT NUMBER: 131:350240
TITLE: sequence of Neospora protein antigens for vaccine development against neosporosis
INVENTOR(S): Brake, David Alan; Madura, Rebecca Anne
PATENT ASSIGNEE(S): Pfizer Products Inc., USA
SOURCE: Eur. Pat. Appl., 59 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 953641	A2	19991103	EP 1999-301746	19990309
EP 953641	A3	20020313		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
EP 1221485	A2	20020710	EP 2002-2959	19990309
EP 1221485	A3	20021002		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, PT, IE, FI, CY				
EP 1221486	A2	20020710	EP 2002-2960	19990309
EP 1221486	A3	20021023		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, PT, IE, FI, CY				
EP 1221487	A2	20020710	EP 2002-2961	19990309

EP 1221487	A3	20021002			
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL					
JP 11332583	A2	19991207	JP 1999-81833		19990325
NZ 334839	A	20000825	NZ 1999-334839		19990325
ZA 9902309	A	20001010	ZA 1999-2309		19990325
AU 9922437	A1	19991007	AU 1999-22437		19990326
CN 1232087	A	19991020	CN 1999-104381		19990326
BR 9902019	A	20000502	BR 1999-2019		19990326
PRIORITY APPLN. INFO.:			US 1998-79389P	P	19980326
			US 1998-112282P	P	19981215
			EP 1999-301746	A3	19990309

AB The present invention provides isolated **polynucleotide** mols. comprising nucleotide sequences encoding GRA1, GRA2, SAG1, MIC1 and MAG1 antigen proteins from Neospora caninum, as well as **recombinant** vectors, transformed host cells, and **recombinantly**-expressed proteins. The present invention further provides a **polynucleotide** mol. comprising the nucleotide sequence of the bidirectional GRA1/MAG1 promoter of N. caninum. The present invention further provides genetic constructs based on the **polynucleotide** mols. of the present invention that are useful in prepg. modified strains of Neospora cells for use in vaccines against neosporosis.

L29 ANSWER 21 OF 26 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER:	1999:198495 HCAPLUS
DOCUMENT NUMBER:	131:54458
TITLE:	**Recombinant respiratory syncytial virus** bearing a **deletion** of either the NS2 or **SH** gene is attenuated in chimpanzees
AUTHOR(S):	Whitehead, Stephen S.; Bukreyev, Alexander; Teng, Michael N.; Firestone, Cai-Yen; St. Claire, Marisa; Elkins, William R.; Collins, Peter L.; Murphy, Brian R.
CORPORATE SOURCE:	Respiratory Viruses Section, Laboratory of Infectious Diseases, National Institute of Allergy and Infectious Diseases, Bethesda, MD, 20892, USA
SOURCE:	Journal of Virology (1999), 73(4), 3438-3442 CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER:	American Society for Microbiology
DOCUMENT TYPE:	Journal
LANGUAGE:	English

AB The NS2 and SH genes of **respiratory syncytial virus** (**RSV**) have been sep. deleted from a **recombinant** wild-type **RSV** strain, A2 (M. N. Teng and P. L. Collins, J. Virol. 73:466-473, 1998; A. Bukreyev et al., J. Virol. 71:8973-8982, 1997; and this study). The resulting viruses, designated rA2.DELTA.NS2 and rA2.DELTA.SH, were administered to chimpanzees to evaluate their levels of attenuation and immunogenicity. **Recombinant** virus rA2.DELTA.NS2 replicated to moderate levels in the upper respiratory tract, was highly attenuated in the lower respiratory tract, and induced significant resistance to challenge with wild-type **RSV**. The replication of rA2.DELTA.SH virus was only moderately reduced in the lower, but not the upper, respiratory tract. However, chimpanzees infected with either virus developed significantly less rhinorrhea than those infected with wild-type **RSV**. These findings demonstrate that a **recombinant RSV mutant** lacking either the NS2 or SH gene is attenuated and indicate that these deletions may be useful as attenuating mutations in new, live **recombinant RSV** vaccine candidates for both

pediatric and elderly populations. The .DELTA.SH mutation was incorporated into a **recombinant** form of the cpts248/404 vaccine candidate, was evaluated for safety in seroneg. chimpanzees, and can now be evaluated as a vaccine for **humans**.
REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 22 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1998:746361 HCAPLUS
DOCUMENT NUMBER: 130:105997
TITLE: Unstable retrovirus **mutants** with acquired transforming activity: rapid changes in the number of repeats of a specific junD **polynucleotide** segment
AUTHOR(S): Ito, Taiji; Kabuyama, Yukihito; Okazaki, Satoshi; Kameda, Takashi; Murakami, Masao; Iba, Hideo
CORPORATE SOURCE: Department of Gene Regulation, Institute of Medical Science, University of Tokyo, Tokyo, 108-8639, Japan
SOURCE: Nucleic Acids Research (1998), 26(21), 4868-4873
CODEN: NARHAD; ISSN: 0305-1048
PUBLISHER: Oxford University Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB We have previously reported that the non-transforming junD (wild type) gene can acquire transforming activity through spontaneous mutations when it is replicated through avian replication-competent retrovirus vectors in chicken embryo fibroblasts. In two of these spontaneous **mutants**, T1 and T2, which were isolated from proviral DNA in the same transformed cell clone, a specific 48 bp **polynucleotide** segment of the junD coding sequence was tandemly repeated three and five times, resp. We report here that the no. of direct repeats in these **mutants** rapidly changes (mostly decreases) in the context of either **RSV**-based replication-competent or MLV-based replication-defective retroviruses, most likely during the process of reverse transcription, while these mutations are stable in the cellular chromosome. We also show that the growth conditions of the infected culture modulate the proportions of polymorphic proviral populations in the infected culture. We finally discuss the possible mol. mechanisms that generate genetic diversity in these amplification **mutants**.
REFERENCE COUNT: 23 THERE ARE 23 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 23 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1998:268362 HCAPLUS
DOCUMENT NUMBER: 128:320547
TITLE: Method of vaccinating infants against infections
INVENTOR(S): Ertl, Hildegund C. J.
PATENT ASSIGNEE(S): Wistar Institute of Anatomy & Biology, USA; Ertl, Hildegund C. J.
SOURCE: PCT Int. Appl., 66 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9817283	A1	19980430	WO 1997-US19509	19971023
W: AU, CA, JP, US				

RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE

AU 9850918	A1	19980515	AU 1998-50918		19971023
US 2001000176	A1	20010405	US 2000-727214		19991130
PRIORITY APPLN. INFO.:			US 1996-29463P	P	19961025
			WO 1997-US19509	W	19971023
			US 1999-284525	A1	19990414

AB A method for overcoming maternal inhibition to a vaccine in a mammalian infant under 1 yr of age, provided by administering to the infant in a suitable pharmaceutical carrier, a **recombinant polynucleotide** sequence (i.e., a **recombinant** virus or DNA vaccine) comprising a sequence encoding an antigen of a pathogenic organism. The **polynucleotide** vector (i.e., virus or DNA vaccine) useful in this method does not naturally cause a pathogenic infection in the species of the mammalian infant to which the vaccine is administered. The vector virus is a replication-defective adenovirus, poxvirus or retrovirus, and the pathogenic organism is rabies virus, **respiratory syncytial virus**, rotavirus, **human** immunodeficiency virus or measles virus. The DNA vaccine is esp. useful for veterinary vaccination on domestic pet or livestock.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L29 ANSWER 24 OF 26 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1997:802696 HCAPLUS

DOCUMENT NUMBER: 128:100868

TITLE: **Respiratory syncytial virus** (**RSV**) SH and G proteins are not essential for viral replication in vitro: clinical evaluation and molecular characterization of a cold-passaged, attenuated **RSV** subgroup B **mutant**

AUTHOR(S): Karron, Ruth A.; Buonagurio, Deborah A.; Georgiu, Alice F.; Whitefield, Stephen S.; Adamus, Jean E.; Clements-Mann, Mary Lou; Harris, Denos O.; Randolph, Valerie B.; Udem, Stephen A.; Murphy, Brian R.; Sidhu, Mohinderjit S.

CORPORATE SOURCE: Cent. Immunization Res., Dep. Intl. Health, Sch. Hygiene Public Health, John Hopkins Univ., Baltimore, MD, 21205, USA

SOURCE: Proceedings of the National Academy of Sciences of the United States of America (1997), 94(25), 13961-13966
CODEN: PNASA6; ISSN: 0027-8424

PUBLISHER: National Academy of Sciences

DOCUMENT TYPE: Journal

LANGUAGE: English

AB A live, cold-passaged (cp) candidate vaccine virus, designated **respiratory syncytial virus** (**RSV**) B1 cp-52/2B5 (cp-52), replicated efficiently in Vero cells, but was over-attenuated for **RSV**-seroneg. infants and children. Sequence anal. of reverse-transcription-PCR-amplified fragments of this **mutant** revealed a large deletion spanning most of the coding sequences for the small hydrophobic (SH) and attachment (G) proteins. Northern blot anal. of cp-52 detected multiple unique read-through mRNAs contg. SH and G sequences, consistent with a **deletion** mutation spanning the **SH**:G gene junction. Immunol. studies confirmed that an intact G glycoprotein was not produced by the cp-52 virus. Nonetheless, cp-52 was infectious and replicated to high titer in tissue culture despite the absence of the viral surface SH and G glycoproteins. Thus, the authors' characterization of this neg.-strand RNA virus identified a novel replication-competent deletion **mutant** lacking

2 of its 3 surface glycoproteins. The requirement of SH and G for efficient replication in vivo suggests that selective deletion of one or both of these **RSV** genes may provide an alternative or additive strategy for developing an optimally attenuated vaccine candidate.

L29 ANSWER 25 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1997:744681 HCAPLUS
DOCUMENT NUMBER: 128:45651
TITLE: **Recombinant respiratory syncytial virus** from which the entire **SH** gene has been **deleted** grows efficiently in cell culture and exhibits site-specific attenuation in the respiratory tract of the mouse
AUTHOR(S): Bukreyev, Alexander; Whitehead, Stephen S.; Murphy, Brian R.; Collins, Peter L.
CORPORATE SOURCE: Laboratory of Infectious Diseases, National Institute Allergy and Infectious Diseases, Bethesda, MD, 20892-0720, USA
SOURCE: Journal of Virology (1997), 71(12), 8973-8982
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The small hydrophobic protein SH of **human respiratory syncytial virus** (**RSV**) is a short transmembrane surface protein of unknown function. A full-length cDNA of **RSV** strain A2 (subgroup A) antigenomic RNA was modified such that the entire SH gene, including the transcription signals and the complete mRNA-encoding sequence, was deleted and replaced by a synthetic intergenic region. This reduced the length of the antigenome by 398 nucleotides and ablated expression of 1 of the 10 **RSV** mRNAs. **Recombinant** virus contg. this engineered deletion was recovered, and the absence of the SH gene was confirmed by reverse transcription in conjunction with PCR. Northern blot anal. of intracellular RNAs and gel electrophoresis of labeled intracellular proteins confirmed the lack of expression of the SH mRNA and protein. The absence of the SH gene did not noticeably affect RNA replication, but two effects on transcription were noted. First, synthesis of the G, F, and M2 mRNAs was increased, presumably due to their being one position closer to the promoter in the gene order. Second, transcription of genes downstream of the engineered site exhibited a steeper gradient of polarity. On monolayers of HEp-2 cells, the SH-minus virus produced syncytia which were at least equiv. in size to those of the wild type and produced plaques which were 70% larger. Furthermore, the SH-minus virus grew somewhat better (up to 12.6-fold) than wild-type **recombinant RSV** in certain cell lines. While the function of the SH protein remains to be detd., it seems to be completely dispensable for growth in tissue culture and fusion function. When inoculated intranasally into mice, the SH-minus virus resembled the wild-type **recombinant** virus in its efficiency of replication in the lungs, whereas it replicated 10-fold less efficiently in the upper respiratory tract. In mice, the SH-minus and wild-type **recombinant** viruses were similarly immunogenic and effective in inducing resistance to virus challenge.

L29 ANSWER 26 OF 26 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1987:191506 HCAPLUS
DOCUMENT NUMBER: 106:191506
TITLE: Interactions of retroviral structural proteins with single-stranded nucleic acids

AUTHOR(S): Karpel, Richard L.; Henderson, Louis E.; Oroszlan, Stephen
CORPORATE SOURCE: Dep. Chem., Univ. Maryland, Catonsville, MD, 21228, USA
SOURCE: Journal of Biological Chemistry (1987), 262(11), 4961-7
CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The interactions of single-stranded polyribonucleotides with murine leukemia virus (MuLV) structural proteins p10, p10' (a p10 **variant**), and Pr65gag, and Rous sarcoma virus (**RSV**) pp12 (a p10 analog were studied). Two quant. assays were used to monitor protein-RNA assocn.: (1) the fluorescence enhancement of polyethenoadenylic acid (poly(.epsilon.A)) upon binding protein, and (2) tryptophan fluorescence quenching upon binding to poly(U). With each assay p10 was shown to bind stoichiometrically to single-stranded RNA, covering a length of nucleic acid chain (occluded site size, n) of .apprx.6 residues. **RSV** pp12 was also shown to bind to poly(.epsilon.A), with n = .apprx.5. Addn. of NaCl to fully titrated MuLV p10-nucleic acid mixts. effected nearly complete restoration of poly(.epsilon.A) or MuLV p10 fluorescence. Under conditions of 0.06M NaCl, p10 bound noncooperatively to poly(.epsilon.A) with an intrinsic assocn. const., K = 2.3 .times. 106 M-1. K And n detd. in this study were shown to relate to Kapp detd. by other methods, by the approxn. Kapp .apprx. NK, where N is the no. of binding sites along the **polynucleotide** chain ((nucleotides/chain)/n). Chem. modifications of the p10 cysteine residues did not alter the affinity for poly(.epsilon.A). The affinity of Pr65gag for poly(.epsilon.A) appears to be higher than that of p10.

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=> d que stat l28
L15        4045 SEA FILE=HCAPLUS ABB=ON  (?RESPIRATORY?(W)?SYNCYTIAL?(W)?VIRUS?
                 OR RSV)
L16        1148 SEA FILE=HCAPLUS ABB=ON  L15 AND (?RECOMB? OR ?VARIANT? OR
                 ?MUTANT?)
L17          11 SEA FILE=HCAPLUS ABB=ON  L16 AND (?DELET?(5A)(?SMALL?(W)?HYDROP
                 HOB? OR SH))
L18          16 SEA FILE=HCAPLUS ABB=ON  L16 AND ?POLYNUCLEOTID?
L19          26 SEA FILE=HCAPLUS ABB=ON  L17 OR L18
L20          19 SEA FILE=HCAPLUS ABB=ON  L19 AND ?HUMAN?
L21           7 SEA FILE=HCAPLUS ABB=ON  L19 AND ?PARAINFLUENZ?
L22          26 SEA FILE=HCAPLUS ABB=ON  L19 OR L20 OR L21
L23          87 SEA L22
L24          67 DUP REMOV L23 (20 DUPLICATES REMOVED)
L25           1 SEA FILE=HCAPLUS ABB=ON  L16 AND ?POLYNUCLEOTID?(5A)(?RESPIRATO
                 RY?(W)?SYNCYTIAL?(W)?VIRUS? OR RSV)
L26          11 SEA L25
L27          11 DUP REMOV L26 (0 DUPLICATES REMOVED)
L28          67 SEA L24 OR L27


=> d ibib abs l24 1-67

L24  ANSWER 1 OF 67  WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER:     2003-278761 [27]   WPIDS
CROSS REFERENCE:      2003-221593 [21]; 2003-221602 [21]
DOC. NO. CPI:         C2003-073023
TITLE:                New expression cassettes and polynucleotides
                      encoding HIV Gag, Nef, Prot, Tat, Rev, Vif, Vpr, Vpu, or
                      Env polypeptides, useful for DNA immunization or
                      generating an immune response against HIV in a subject.
DERWENT CLASS:        B04 D16
INVENTOR(S):          BARNETT, S W; LIAN, Y; ZUR MEGEDE, J
PATENT ASSIGNEE(S):   (CHIR) CHIRON CORP
COUNTRY COUNT:        88
PATENT INFORMATION:

     PATENT NO   KIND DATE     WEEK      LA   PG
     -------------------------------------------
     WO 2003020876 A2 20030313 (200327)* EN  214
        RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR IE IT KE LS LU
            MC MW MZ NL OA PT SD SE SK SL SZ TR TZ UG ZM ZW
         W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD
            GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV
            MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT
            UA UG US UZ VN YU ZA ZW

APPLICATION DETAILS:

     PATENT NO   KIND                  APPLICATION      DATE
     ------------------------------------------------------------
     WO 2003020876 A2                  WO 2002-US21342  20020705


PRIORITY APPLN. INFO: US 2002-349728P  20020116; US 2001-316860P
                      20010831
AN   2003-278761 [27]   WPIDS
CR   2003-221593 [21]; 2003-221602 [21]
AB   WO2003020876 A UPAB: 20030429
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NOVELTY - An expression cassette comprising a **polynucleotide** sequence encoding a polypeptide including an HIV Gag, Nef, Prot, Tat, Rev, Vif, Vpr, Vpu, or Env polypeptide, is new.

DETAILED DESCRIPTION - The **polynucleotide** sequence encoding:

(a) the Gag polypeptide comprises a sequence having at least 90% sequence identity to any one of 5 sequences of 4773-5274 base pairs (bp), a sequence having at least 98% sequence identity to any one of 4 sequences (e.g. 4883 bp), or a sequence having at least 95% sequence identity to a sequence of 3004 bp, all fully defined in the specification;

(b) the Nef polypeptide comprises a sequence having at least 90% sequence identity to any one of 6 fully defined sequences of 570-3735 bp given in the specification;

(c) the Prot polypeptide comprises a sequence having at least 98% sequence identity to a fully defined sequence of 2262 bp given in the specification;

(d) the Tat polypeptide comprises a sequence having at least 90% sequence identity to any one of 8 fully defined sequences of 1281-5283 bp given in the specification;

(e) the Rev polypeptide comprises a sequence having at least 90% sequence identity to a fully defined sequence of 348 bp given in the specification;

(f) the Vif polypeptide comprises a sequence having at least 90% sequence identity to at least 30 contiguous base pairs of a fully defined sequence of 576 bp given in the specification;

(g) the Vpr polypeptide comprises a sequence having at least 90% sequence identity to at least 20 contiguous base pairs of a fully defined sequence of 291 bp given in the specification;

(h) the Vpu polypeptide comprises a sequence having at least 90% sequence identity to at least 20 contiguous base pairs of a fully defined sequence of 243 bp given in the specification; or

(i) the Env polypeptide comprises a sequence having at least 90% sequence identity to any one of 4 fully defined sequences each comprising 2007 bp given in the specification.

INDEPENDENT CLAIMS are included for the following:

(1) a **recombinant** expression system for use in a selected host cell comprising any one of the expression cassettes cited above, where the **polynucleotide** sequence is operably linked to control elements compatible with expression in the selected host cell;

(2) a cell comprising the expression cassette, where the **polynucleotide** sequence is operably linked to control elements compatible with expression in the selected cell;

(3) a method for producing a polypeptide including HIV Gag polypeptide sequences, comprising incubating the cells of (2) to produce the polypeptide;

(4) a gene delivery vector for use in a mammalian subject, where the vector comprises the expression cassette, and where the **polynucleotide** sequence is operably linked to control elements compatible with expression in the subject;

(5) a method of DNA immunization of a subject comprising introducing the gene delivery vector into the subject under conditions that are compatible with expression of the expression cassette in the subject; and

(6) a method of generating an immune response in a subject comprising transfecting cells of the subject with the gene delivery vector under conditions that permit the expression of the **polynucleotide** and production of the polypeptide to elicit an immunological response to the polypeptide.

ACTIVITY - Anti-HIV.

MECHANISM OF ACTION - Vaccine. Rabbits were immunized intramuscularly, mucosally or intradermally with plasmid DNAs encoding the

HIV proteins. The nucleic acid immunizations were followed by protein boosting after the initial immunization. Constructs comprising the synthetic HIV polypeptide-encoding **polynucleotides** were highly immunogenic and generated substantial antigen binding antibody responses after only two immunizations.

USE - The expression cassettes, HIV polypeptides and **polynucleotides** encoding the HIV polypeptides are useful for DNA immunization or generating an immune response against HIV in a subject. The **polynucleotides** are also useful for generating packaging cell lines or producing the HIV polypeptides.

Dwg.0/68

L24 ANSWER 2 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2003-300736 [29] WPIDS
DOC. NO. CPI: C2003-078428
TITLE: Use of a composition comprising **recombinant** adeno-associated virus virions for delivering glial cell line-derived neutrophic factor, useful in treating a **human** subject with preexisting neuronal damage, e.g. Parkinson's disease.
DERWENT CLASS: B04 D16
INVENTOR(S): MURAMATSU, S; OZAWA, K
PATENT ASSIGNEE(S): (MURA-I) MURAMATSU S; (OZAW-I) OZAWA K
COUNTRY COUNT: 101
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003018821	A2	20030306	(200329)*	EN	38

RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SK SL SZ TR TZ UG ZM ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003018821	A2	WO 2002-JP8761	20020829

PRIORITY APPLN. INFO: US 2001-315838P 20010829
AN 2003-300736 [29] WPIDS
AB WO2003018821 A UPAB: 20030505

NOVELTY - A composition comprising **recombinant** adeno-associated virus (AAV) virions is used for treating a mammalian subject with preexisting neuronal damage by administration into the subject's central nervous system. The virions comprise a **polynucleotide** encoding a glial cell line-derived neutrotrophic factor (GDNF) polypeptide operably linked to expression control elements that comprise a promoter.

ACTIVITY - Nootropic; Neuroprotective; Antiparkinsonian. No biological data given.

MECHANISM OF ACTION - Gene therapy.

Behavioural recovery after intrastriatal injection of AAV-GDNFflag. Four weeks after 6-OHDA administration, all rats in the three groups (AAV-GDNFflag, n=20; AAV-LacZ, n=16; and vehicle, n=8) demonstrated a similar degree of impairment both in the apomorphine-induced rotation

tests and in the cylinder tests, suggesting equivalent levels of dopaminergic (DA) depletion. A decrease in the rotations was observed in rats receiving AAV-GDNFflag, beginning at 4 weeks after the vector injection. The decrease proceeded gradually and persisted through the experiment (20 weeks). In contrast, rats in the AAV-LacZ or vehicle injection group demonstrated a fairly stable rotation rate during the same period.

USE - The composition is useful in treating **human** subjects with preexisting neuronal damage that comprises moderate to extensive nigrostriatal dopaminergic (DA) denervation (claimed). It uses AAV-based gene delivery systems for delivering GDNF to subjects with neurodegenerative conditions such as Parkinson's disease.

Dwg.0/13

L24 ANSWER 3 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2003-221602 [21] WPIDS
CROSS REFERENCE: 2003-221593 [21]; 2003-278761 [27]
DOC. NO. CPI: C2003-056373
TITLE: New synthetic **polynucleotides** encoding antigenic HIV type B and/or type C polypeptides, useful as immunogenic compositions or vaccines for generating humoral or cellular immune responses against HIV in a subject, especially **humans**.
DERWENT CLASS: B04 C06 D16
INVENTOR(S): BARNETT, S W; LIAN, Y; ZUR MEGEDE, J
PATENT ASSIGNEE(S): (CHIR) CHIRON CORP
COUNTRY COUNT: 88
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003004657	A1	20030116	(200321)*	EN	256

RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SK SL SZ TR TZ UG ZM ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZA ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003004657	A1	WO 2002-US21421	20020705

PRIORITY APPLN. INFO: US 2002-349871P 20020116; US 2001-303192P 20010705; US 2001-316860P 20010831; US 2002-349728P 20020116; US 2002-349793P 20020116
AN 2003-221602 [21] WPIDS
CR 2003-221593 [21]; 2003-278761 [27]
AB WO2003004657 A UPAB: 20030429
NOVELTY - A synthetic **polynucleotide** encoding 2 or more immunogenic HIV polypeptides, where at least 2 of the polypeptides are derived from different HIV subtypes, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:

(1) an expression cassette comprising the synthetic **polynucleotide**;

(2) a **recombinant** expression system for use in a host cell comprising the expression cassette, where the **polynucleotide**

sequence is operably linked to control elements compatible with expression in the host cell;

(3) a cell comprising the expression cassette, where the **polynucleotide** sequence is operably linked to control elements compatible with expression in the cell;

(4) a gene delivery vector for use in a mammalian subject, where the vector comprises the expression cassette, and the **polynucleotide** sequence is operably linked to control elements compatible with expression in the subject;

(5) a method for producing a polypeptide including 2 or more HIV polypeptides from different subtypes by incubating the cells of (3) to produce the polypeptide;

(6) a method of DNA immunization of a subject by introducing the gene delivery vector into a subject under conditions that are compatible with expression of the expression cassette in the subject; and

(7) a method of generating an immune response in a subject by transfecting the cells of the subject with the gene delivery vector, under conditions that permit the expression of the **polynucleotide** and the production of the polypeptide, thus eliciting an immunological response to the polypeptide.

ACTIVITY - Anti-HIV.

MECHANISM OF ACTION - Vaccine; Gene Therapy.

No biological data given.

USE - The **polynucleotide** is useful for immunization, generation of packaging cell lines, or production of HIV polypeptides. The **polynucleotide** and its encoded proteins are useful as immunogenic compositions or vaccines for generating humoral or cellular immune responses against HIV in a subject, or for inducing neutralizing antibodies against HIV. The gene delivery vector comprising the **polynucleotide** is also useful for DNA immunization of, or for generating an immune response (e.g. a humoral or cellular immune response) in, a subject such as a mammal, particularly a **human** (claimed).

Dwg.0/96

L24 ANSWER 4 OF 67 WPIDS (C) 2003 THOMSON DERWENT

ACCESSION NUMBER: 2003-221593 [21] WPIDS

CROSS REFERENCE: 2003-221602 [21]; 2003-278761 [27]

DOC. NO. CPI: C2003-056364

TITLE: New expression cassette comprising a **polynucleotide** sequence encoding a polypeptide including an HIV Gag, Env, Int, Nef, p15RnaseH, Pol, Tat, Prot, or Rev polypeptide, useful for immunization, or generating packaging cell lines.

DERWENT CLASS: B04 D16

INVENTOR(S): BARNETT, S W; ENGELBRECHT, S; LIAN, Y; VAN RENSBURG, E J; ZUR MEGEDE, J

PATENT ASSIGNEE(S): (CHIR) CHIRON CORP; (UYST-N) UNIV STELLENBOSCH

COUNTRY COUNT: 100

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003004620	A2	20030116	(200321)*	EN	301

RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SK SL SZ TR TZ UG ZM ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG US UZ VN YU ZA ZM ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003004620 A2		WO 2002-US21420	20020705

PRIORITY APPLN. INFO: US 2002-349871P 20020116; US 2001-303192P 20010705; US 2001-316860P 20010831

AN 2003-221593 [21] WPIDS

CR 2003-221602 [21]; 2003-278761 [27]

AB WO2003004620 A UPAB: 20030429

NOVELTY - An expression cassette (I) comprising a **polynucleotide** sequence encoding a polypeptide including an HIV Gag, Env, Int, Nef, p15RnaseH, Pol, Tat, Prot, or Rev polypeptide, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) a **recombinant** expression system (II) for use in a selected host cell comprising the expression cassette, where the **polynucleotide** sequence is operably linked to control elements compatible with expression in the selected host cell;

(2) a cell (III) comprising (II);

(3) producing (M1) a polypeptide including HIV Gag polypeptide sequences by incubating (III) under conditions to produce the polypeptide;

(4) a gene delivery vector (IV) for use in mammalian subject comprising (I);

(5) DNA immunization (M2) of a subject by introducing the gene delivery vector into a subject under conditions that are compatible with expression of the expression cassette; and

(6) generating (M3) an immune response in a subject by transfecting cells of the subject the gene delivery vector under conditions to permit the expression of the **polynucleotide** and production of the polypeptide, therefore eliciting an immunological response to the polypeptide.

ACTIVITY - None given.

No biological data given.

MECHANISM OF ACTION - Gene therapy; Vaccine.

USE - The expression cassette is useful for immunization, generating packaging cell lines and producing HIV polypeptides.

Dwg.0/123

L24 ANSWER 5 OF 67 MEDLINE DUPLICATE 1

ACCESSION NUMBER: 2003147801 IN-PROCESS

DOCUMENT NUMBER: 22550062 PubMed ID: 12663767

TITLE: **Respiratory Syncytial Virus** (**RSV**) Fusion Protein Subunit F2, Not Attachment Protein G, Determines the Specificity of **RSV** Infection.

AUTHOR: Schlender Jorg; Zimmer Gert; Herrler Georg; Conzelmann Karl-Klaus

CORPORATE SOURCE: Max von Pettenkofer Institute and Gene Center, Ludwig-Maximilians-University Munich, D-81377 Munich. Institute of Virology, School of Veterinary Medicine, D-30559 Hannover, Germany.

SOURCE: JOURNAL OF VIROLOGY, (2003 Apr) 77 (8) 4609-16. Journal code: 0113724. ISSN: 0022-538X.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20030331
Last Updated on STN: 20030331

AB **Human respiratory syncytial virus** (HRSV) and bovine **RSV** (BRSV) infect **human** beings and cattle in a species-specific manner. We have here analyzed the contribution of **RSV** envelope proteins to species-specific entry into cells. In contrast to permanent cell lines, primary cells of **human** or bovine origin, including differentiated respiratory epithelia, peripheral blood lymphocytes, and macrophages, showed a pronounced species-specific permissivity for HRSV and BRSV infection, respectively. **Recombinant** BRSV **deletion mutants** lacking either the **small hydrophobic** (SH) protein gene or both SH and the attachment glycoprotein (G) gene retained their specificity for bovine cells, whereas corresponding **mutants** carrying the HRSV F gene specifically infected **human** cells. To further narrow the responsible region of F, two reciprocal chimeric F constructs were assembled from BRSV and HRSV F1 and F2 subunits. The specificity of **recombinant RSV** carrying only the chimeric F proteins strictly correlated with the origin of the membrane-distal F2 domain. A contribution of G to the specificity of entry could be excluded after reintroduction of BRSV or HRSV G. Virus with F1 and G from BRSV and with only F2 from HRSV specifically infected **human** cells, whereas virus expressing F1 and G from HRSV and F2 from BRSV specifically infected bovine cells. The introduction of G enhanced the infectivities of both chimeric viruses to equal degrees. Thus, the role of the nominal attachment protein G is confined to facilitating infection in a non-species-specific manner, most probably by binding to cell surface glycosaminoglycans. The identification of the F2 subunit as the determinant of **RSV** host cell specificity facilitates identification of virus receptors and should allow for development of reagents specifically interfering with **RSV** entry.

L24 ANSWER 6 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2003-120788 [11] WPIDS
DOC. NO. CPI: C2003-031353
TITLE: Chimeric transmembrane protein for manufacturing a medicament for treating or preventing viral infection, comprises an extracellular domain capable of binding a virus and an intracellular internalization signal.
DERWENT CLASS: B04 D16
INVENTOR(S): GOH, P Y; HONG, W J; LIM, S G; LIM, S P; TAN, Y H; TAN, Y J
PATENT ASSIGNEE(S): (MOLE-N) INST MOLECULAR & CELL BIOLOGY
COUNTRY COUNT: 100
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002094874	A2	20021128	(200311)*	EN	63

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG US UZ VN YU ZA ZM ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002094874 A2		WO 2002-CA762	20020524

PRIORITY APPLN. INFO: GB 2001-12652 20010524
AN 2003-120788 [11] WPIDS
AB WO 200294874 A UPAB: 20030214
NOVELTY - A chimeric transmembrane protein comprising:
(a) an extracellular domain capable of binding a virus; and
(b) an intracellular internalization signal, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:
(1) a **polynucleotide** encoding the protein;
(2) a vector comprising the **polynucleotide**;
(3) a cell comprising the protein;
(4) producing the cell comprising transfecting a cell with (1) or (2) and maintaining the cell under conditions suitable for obtaining expression of the protein;
(5) a transgenic non-**human** animal comprising the cell; and
(6) identifying an anti-viral agent comprising:
(a) providing (3) or (5);
(b) contacting (3) or (5) with a test agent; and
(c) monitoring viral infection;
(7) identifying an antiviral vaccine or agent capable of preventing or inhibiting viral infection comprising:
(a) providing (3) or (5);
(b) contacting (3) or (5) with a test agent;
(c) contacting (3) or (5) with a virus capable of binding to the protein; and
(d) determining whether the test agent prevents or limits viral infection; and
(8) an antiviral agent or vaccine identified by (6), or (7).
ACTIVITY - Virucide. No biological data is given.
MECHANISM OF ACTION - Vaccine; Gene therapy.
USE - The protein is useful for identifying an antiviral agent or a vaccine, which are used for manufacturing a medicament for treating or preventing viral infection. The viral infections are herpes C virus (HCV), **human** immunodeficiency virus (HIV), herpes B virus (HBV), rous sarcoma virus (**RSV**), influenza virus, herpes simplex virus, rabies virus, coxsackie virus, or rhinovirus (claimed).
Dwg.0/13

L24 ANSWER 7 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2002-508507 [54] WPIDS
DOC. NO. CPI: C2002-144574
TITLE: Isolated infectious **respiratory syncytial virus** particle, useful as a vaccine, has an attenuated phenotype comprising the viral genome that has a heterologous sequence encoding a G and F protein and a mutation in the M2-2 gene.
DERWENT CLASS: B04 D16
INVENTOR(S): BRYANT, M; JIN, H; LI, S; TANG, R
PATENT ASSIGNEE(S): (AVIR-N) AVIRON INC
COUNTRY COUNT: 98
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002044334	A2	20020606	(200254)*	EN	150

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ
NL OA PT SD SE SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK
DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR
KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT
RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW
AU 2002036522 A 20020611 (200264)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002044334 A2		WO 2001-US44819	20011128
AU 2002036522 A		AU 2002-36522	20011128

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2002036522 A		Based on	WO 200244334

PRIORITY APPLN. INFO: US 2000-724416 20001128
AN 2002-508507 [54] WPIDS
AB WO 200244334 A UPAB: 20021031
NOVELTY - An isolated infectious **respiratory syncytial virus** (**RSV**) particle (I) with an attenuated phenotype comprising an **RSV** antigenome or genome, where the genome or antigenome has a heterologous sequence encoding a G and F protein, and a mutation in the M2-2 gene, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:
(1) an isolated cDNA (II) encoding (I);
(2) and a vaccine (III) or a pharmaceutical composition (IV) comprising (I) or (II), where (II) or the genome of (I) contains the reverse complement of an mRNA coding sequence operatively linked to a polymerase binding site of a **RSV**, where the mRNA coding sequence contains a deletion in the M2-2 gene and a heterologous sequence encoding the F and G protein, and a pharmaceutically acceptable carrier.
ACTIVITY - None given.
MECHANISM OF ACTION - Vaccine (claimed). No biological data is given.
USE - (I) is useful as expression vector or vaccine.
Dwg.0/28

L24 ANSWER 8 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2002-599372 [64] WPIDS
DOC. NO. CPI: C2002-169205
TITLE: Facilitating production of a protein for analyzing, designing and/or modifying an agent that can interact with a viral F protein, comprises expressing a nucleic acid optimized for expression of the protein, using a eukaryotic cell.
DERWENT CLASS: B04 D16
INVENTOR(S): MASON, A J; TUCKER, S P; YOUNG, P R
PATENT ASSIGNEE(S): (BIOT-N) BIOTA SCI MANAGEMENT PTY LTD
COUNTRY COUNT: 99
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002042326	A1	20020530	(200264)*	EN	367

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ
NL OA PT SD SE SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK
DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR
KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT
RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZM ZW
AU 2002023275 A 20020603 (200264)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002042326 A1		WO 2001-AU1517	20011122
AU 2002023275 A		AU 2002-23275	20011122

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2002023275 A	Based on	WO 200242326

PRIORITY APPLN. INFO: US 2000-252767P 20001122
AN 2002-599372 [64] WPIDS
AB WO 200242326 A UPAB: 20021007
NOVELTY - Facilitating (M1) production of a protein or its derivative (I) from a negative sense single stranded RNA virus, by expressing a nucleic acid molecule (NAM) encoding (I) in a host cell, where the nucleotide sequence of NAM is optimized for expression by a eukaryotic cell.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an optimized NAM or its derivative, equivalent, analog or mimetic (II);

(2) a protein molecule (I) encoded (II);

(3) regulating (M2) the functional activity of a viral F protein, where the protein in its non-fully functional form comprises an F2 portion linked, bound or otherwise associated with an F1 portion, and where the F2 portion comprises an intervening peptide sequence, by modulating cleavage of the intervening peptide sequence where excision of a portion of the intervening sequence from the non-fully functional form of the protein up-regulates F protein functional activity;

(4) detecting (M3) an agent capable of regulating the functional activity of a viral F protein or its derivative by contacting an eukaryotic cell expressing an optimized NAM with a putative modulatory agent and detecting an altered expression phenotype and/or functional activity;

(5) an agent (III) capable of interacting with a viral F protein and modulating a functional activity associated with the viral protein;

(6) a viral F protein **variant** (IV) comprising a mutation in the intervening peptide sequence, where the **variant** exhibits modulated functional activity relative to wild-type F protein or its derivative, homolog, analog, chemical equivalent or mimetic;

(7) a **recombinant** viral construct (RVC) comprising NAM, where the **recombinant** viral construct is a effective in inducing, enhancing or otherwise stimulating an immune response to the F protein; and

(8) a vaccine comprising RVC.

ACTIVITY - Virucide.

MECHANISM OF ACTION - Vaccine (claimed); Gene therapy. No biological data is given.

USE - (I), especially F protein, is useful for analyzing, designing

and/or modifying an agent capable of interacting with a viral F protein or its derivative and modulating a functional activity associated with the protein, by contacting (I) with a putative agent and assessing the degree of interactive complementarity of the agent with the protein (I). An optimized NAM or its derivative, equivalent, analog or mimetic (II), an agent (III) capable of interacting with a viral F protein and modulating a functional activity associated with the viral protein, or an agent identified using (I) is useful in the manufacture of a medicament utilized in the therapeutic and/or prophylactic treatment of conditions characterized by infection with a negative sense single stranded RNA virus, and for modulating a functional activity associated with a viral F protein in a subject, preferably a mammal, especially a **human**, where the functional activity is F protein mediated host cell virion fusion and/or virion budding and the modulating is down regulation (all claimed).
Dwg.0/8

L24 ANSWER 9 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2002-454551 [48] WPIDS
DOC. NO. CPI: C2002-129237
TITLE: Novel **human** cytomegalovirus Intron A fragment for use in expression constructs, lacks full-length Intron A sequence, and enhance expression levels when present in expression constructs.
DERWENT CLASS: B04 D16
INVENTOR(S): SELBY, M; THUDIUM, K; ULMER, J
PATENT ASSIGNEE(S): (CHIR) CHIRON CORP
COUNTRY COUNT: 96
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002031137	A2	20020418	(200248)*	EN	44

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2002011710 A 20020422 (200254)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002031137	A2	WO 2001-US32050	20011012
AU 2002011710	A	AU 2002-11710	20011012

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2002011710	A Based on	WO 200231137

PRIORITY APPLN. INFO: US 2000-240502P 20001013
AN 2002-454551 [48] WPIDS
AB WO 200231137 A UPAB: 20020730
NOVELTY - A **human** cytomegalovirus (hCMV) Intron A fragment (I) that lacks full-length Intron A sequence (II), is new. When (I) is present in expression construct (C), the (C) achieves expression levels greater

than those levels achieved by a corresponding (C) that completely lacks (II) or equal to, or greater than, those levels achieved by (C) that includes a corresponding intact, full-length (II).

DETAILED DESCRIPTION - A **human** cytomegalovirus (hCMV) Intron A fragment (I) (derived from intron A sequence of CMV immediate-early enhancer/promoter region), where (I) lacks the full-length (II) and comprises a sequence of nucleotides having at least 75 % sequence identity to the contiguous sequence of nucleotides found at positions:

(a) 1-25, inclusive, of a fully defined cytomegalovirus immediate-early region (CMV IE)1 Intron A from hCMV strain Towne sequence of 838 nucleotides (S1), given in specification; and

(b) 775-820, inclusive, of (S1), where when (I) is present in (C), the (C) achieves expression levels greater than those levels achieved by a corresponding construct that completely lacks an Intron A sequence, or equal to, or greater than, those levels achieved by (C) that includes a corresponding intact, full-length (II).

Optionally, (I) comprises a sequence of nucleotides having at least about 75% sequence identity to the contiguous sequence of nucleotides found at positions 1-51, inclusive, of (S1), and 741-820, inclusive of (S1), where when (I) is present in (C), the (C) achieves expression levels greater than those levels achieved by a corresponding construct that completely lacks an Intron A sequence, or equal to, or greater than, those levels achieved by (C) that includes a corresponding intact, full-length (II).

INDEPENDENT CLAIMS are also included for the following:

(1) a **recombinant** expression construct (III) effective in directing transcription of a selected coding sequence, comprising a coding sequence, control elements that are operably linked to the coding sequence, where the control elements comprise (I), where the coding sequence can be transcribed and translated in a host cell;

(2) a host cell (IV) comprising (III); and

(3) a **polynucleotide** comprising a 127 nucleotide optimized rabbit beta -globin gene sequence (S6), given in the specification.

ACTIVITY - Cytostatic.

MECHANISM OF ACTION - Gene therapy.

In order to test the ability of the Intron A fragments to direct transcription in vivo, Balb/c mice in groups of 6 animals were immunized once bilaterally in the tibialis anterior muscle with 5 micro g of naked vector DNA per injection site. Three- and six-week post-immunization bleeds were analyzed by enzyme linked immunosorbent assay (ELISA) for anti-**human** immunodeficiency virus (HIV) p55 gag antibody as described in Zur Megede et al., J.Virol (2000) 74:2628-2635. Variable immunogenicities were seen after a single immunization. Significantly, the pCON3 vector which deleted 85 % of Intron A yielded higher geometric mean titers than the parent pCMVkm2.GAGmod.SF2 vector. At three weeks post-immunization, the titer was twice that of parent vector though this fell off by six weeks post-injection.

USE - (III) is also useful for producing a **recombinant** polypeptide which involves introducing (III) into a host cell and causing expression of the coding sequence of the expression construct to produce the **recombinant** polypeptide. (IV) is useful for producing a **recombinant** polypeptide which involves culturing a population of (IV), under conditions where the coding sequence of the **recombinant** expression construct is expressed, thereby producing the **recombinant** polypeptide. (All claimed). (I) is useful in expression constructs to express a wide variety of substances including peptides. (I) is also useful for producing proteins useful for treating a variety of malignant cancers, and for producing proteins useful for prevention, treatment and/or diagnosis of a wide variety of diseases. (III) is used in nucleic acid immunization and gene therapy.

ADVANTAGE - When (I) is present in a (C), the (C) achieves expression levels at least two-fold (most preferably, fifty-fold) greater than those levels achieved by a corresponding construct that completely lacks (II) (claimed). (I) allows for creation of highly efficient expression system for the production of **recombinant** proteins in therapeutically useful quantities both in vitro and in vivo. The use of (I) reduces the overall plasmid size for expression of particular coding sequence, and thus is desirable when larger coding sequences and/or viral vectors with limited ability to package large genes are used. A decrease in overall size of the constructs effectively enhances efficiency of expression. (I) retains the ability to enhance expression levels even when present in expression constructs, and these high levels of expression provide for immune responses that are comparable to or even better than, that induced by the parent vector. (III) allows for production of desired protein in an authentic configuration, with authentic post-translation modifications, in a relatively pure form and in economically useful amounts.
Dwg.0/7

L24 ANSWER 10 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2002-154920 [20] WPIDS
DOC. NO. CPI: C2002-048494
TITLE: New **polynucleotides** encoding antigenic HIV Type C polypeptides, useful in applications including DNA immunization or generation of packaging cell lines, particularly in gene therapy.
DERWENT CLASS: B04 D16
INVENTOR(S): BARNETT, S W; ENGELBRECHT, S; VAN RENSBURG, E J; ZUR MEGEDE, J
PATENT ASSIGNEE(S): (CHIR) CHIRON CORP; (UYST-N) UNIV STELLENBOSCH
COUNTRY COUNT: 96
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002004493	A2	20020117	(200220)*	EN	233
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2001073179	A	20020121	(200234)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002004493	A2	WO 2001-US21241	20010705
AU 2001073179	A	AU 2001-73179	20010705

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001073179	A Based on	WO 200204493

PRIORITY APPLN. INFO: US 2000-610313 20000705
AN 2002-154920 [20] WPIDS
AB WO 200204493 A UPAB: 20020402
NOVELTY - New expression cassettes comprise a **polynucleotide**

sequence encoding a polypeptide comprising immunogenic HIV type C polypeptides. The expression cassettes comprise any of the sequences encoding Gag, Pol, Vif, Vpr, Tat, Rev, Vpu, Env or Nef, which are fully defined in the specification.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) **polynucleotides** comprising the sequence comprising 9781, 9556 or 2604 base pairs (bp) fully defined in the specification;

(2) a cell comprising any of the expression cassettes, where the **polynucleotide** sequence further comprises control elements compatible with expression in the selected cell;

(3) compositions for generating an immunological response comprising any of the expression cassettes;

(4) a method of immunization of a subject comprising introducing the composition into the subject under conditions that is compatible with expression of the expression cassette in the subject;

(5) a method of generating an immune response in a subject comprising:

(a) providing the expression cassette;

(b) expressing the polypeptide in a suitable host cell;

(c) isolating the polypeptide; and

(d) administering the polypeptide to the subject in an amount sufficient to elicit an immune response; and

(6) a method of generating an immune response in a subject comprising introducing into cells of the subject the expression cassette under conditions that permit the expression of the **polynucleotide** and production of the polypeptide, thereby eliciting an immunological response to the polypeptide.

ACTIVITY - Immunostimulant. No biodata is given in the source material.

MECHANISM OF ACTION - Gene therapy.

USE - The **polynucleotides** are useful in applications including DNA immunization, generation of packaging cell lines, and production of HIV Type C proteins. The **polynucleotides** are particularly useful in gene therapy and DNA immunization applications.

Dwg.0/105

L24 ANSWER 11 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2002-216911 [27] WPIDS
DOC. NO. NON-CPI: N2002-166275
DOC. NO. CPI: C2002-066288
TITLE: Novel bovine **respiratory syncytial virus** useful for preparing vaccines for use in vaccinating cattle against infection by the virus, is incapable of expressing a functional G-protein due to a mutation.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): BUCHHOLZ, U J; SCHMIDT, U
PATENT ASSIGNEE(S): (ALKU) AKZO NOBEL NV
COUNTRY COUNT: 88
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002002752	A1	20020110	(200227)*	EN	36

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW

W: AE AG AL AU BA BB BG BR BZ CA CN CO CR CU CZ DM DZ EE GD GE HR HU ID IL IN IS JP KP KR LC LK LR LT LV MA MG MK MN MX MZ NO NZ PL RO RU SG SI SK SL TR TT UA US UZ VN YU ZA

AU 2001063896 A 20020114 (200237)
EP 1297110 A1 20030402 (200325) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002002752	A1	WO 2001-EP5085	20010502
AU 2001063896	A	AU 2001-63896	20010502
EP 1297110	A1	EP 2001-938170	20010502
		WO 2001-EP5085	20010502

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2001063896	A	Based on	WO 200202752
EP 1297110	A1	Based on	WO 200202752

PRIORITY APPLN. INFO: EP 2000-202183 20000623
AN 2002-216911 [27] WPIDS
AB WO 200202752 A UPAB: 20020429
NOVELTY - A bovine **respiratory syncytial virus** (BRSV) (I) incapable of expressing a functional G-protein due to a mutation in the gene encoding the protein, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a vaccine (II) for protecting cattle against BRSV-infection comprising (I) and a carrier;

(2) diagnostic test kit (III) for discriminating bovine respiratory syncytial field-virus infected animals from animals vaccinated with (II), comprising a G protein or antibodies against G protein;

(3) discriminating (M1) bovine respiratory syncytial field-virus infected animals from animals vaccinated with (II), involves incubating a body fluid or tissue of an animal to be tested with G protein (or antibodies against purified G protein) and optionally, SH protein (or antibodies against SH protein); and

(4) preparing (II) for protecting cattle against BRSV infection involves admixing (I) and a carrier.

ACTIVITY - Virucide; antibacterial.

MECHANISM OF ACTION - Vaccine.

The biological effect of **recombinant** bovine **respiratory syncytial virus** (rBRSV) with a deletion of glycoprotein G (rBRSV Delta G) was tested in calves. Eleven conventionally reared calves were allotted to three groups. Group I (mock vaccination) consisted of three calves, groups II (vaccination with rBRSV Delta G) and group III (vaccination with or rBRSV) consisted of four calves each. In group I, each calf received a mock vaccination consisting of 8 ml of Madin-Darby bovine kidney (MDBK) cell culture suspension intranasally. Animals of group II were vaccinated intranasally with 8 multiply 109 plaque forming units (PFU) or rBRSV. Six weeks after immunization, the calves were challenged with low-passaged wild-type BRSV, strain CA-1 via aerosol. After the challenge, clinical examinations (body temperature, heart rate, breathing rate, coughing and abnormal lung sounds, nasal or ocular discharge depression) blood samples were taken on day eight after challenge. On day eight after the challenge, the calves were necropsied. After challenge, the calves of the mock-immunized group showed mild to severe clinical disease. The calves of the rBRSV Delta G

group showed post-challenge only mild coughing, and for three of four animals a slight raise of rectal temperatures was observed. In the rBRSV group, only one calf showed an increase in rectal temperature, and rare coughing was seen in two calves. After intranasal vaccination, the sera obtained from the calves of rBRSV and the rBRSV Delta G groups showed an increase in neutralizing activity. In the group immunized with rBRSV, the ND(50) titers increased up to values between 32 and 256, with an increase resembling that occurring after natural infections. The ND(50) titers of the rBRSV Delta G-group stayed on a moderate level of 8 to 24 until day 35 after vaccination. After challenge, the animals in the rBRSV Delta G-group showed a four- to sixfold increase in the ND(50) eight days after BRSV exposure, whereas in the rBRSV group, a slight increase in neutralizing activity was found only in one out of four calves.

USE - (I) is useful for preparing vaccines (claimed) which are useful for vaccinating cattle against BRSV infection, and additionally bovine rotavirus, bovine viral diarrhea virus, **parainfluenza** type 3 virus, bovine paramyxovirus, bovine herpesvirus, foot and mouth disease virus or Pasteurella haemolytica.

(M1) is also useful for discriminating bovine respiratory syncytial field-virus infected animals from animals vaccinated with (II) (claimed). Dwg.0/6

L24 ANSWER 12 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2002-130895 [17] WPIDS
DOC. NO. CPI: C2002-040273
TITLE: Producing **recombinant** canine distemper virus (CDV), useful in gene therapy, comprises transfecting cell with transcription vector containing nucleic acid encoding CDV genome or antigenome.
DERWENT CLASS: B04 C06 D16
INVENTOR(S): KOVACS, G R; PARKS, C L; SIDHU, M S; UDEM, S A; WALPITA, P
PATENT ASSIGNEE(S): (AMCY) AMERICAN CYANAMID CO
COUNTRY COUNT: 97
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002000883	A2	20020103	(200217)*	EN	109

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

AU 2001071423	A	20020108	(200235)		
EP 1303613	A2	20030423	(200329)	EN	

R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002000883	A2	WO 2001-US20157	20010622
AU 2001071423	A	AU 2001-71423	20010622
EP 1303613	A2	EP 2001-950430	20010622
		WO 2001-US20157	20010622

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2001071423	A	Based on	WO 200200883
EP 1303613	A2	Based on	WO 200200883

PRIORITY APPLN. INFO: US 2000-213698P 20000623
AN 2002-130895 [17] WPIDS
AB WO 200200883 A UPAB: 20020313
NOVELTY - Producing a **recombinant** canine distemper virus (CDV) comprising transforming/transfecting a cell with a composition containing:
(a) a transcription vector containing an isolated nucleic acid molecule encoding a genome/antigenome of CDV, or its **variant** sequence; and
(b) an expression vector with an isolated nucleic acid molecule encoding the trans-acting proteins (N, P and L) for encapsidation, transcription and replication, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:
(1) a **recombinant** CDV prepared from the method defined above;
(2) compositions comprising a CDV prepared from the method or a **recombinantly** produced CDV, and a pharmaceutical carrier;
(3) an immunogenic composition comprising an isolated, **recombinantly** produced CDV and a physiological carrier;
(4) immunizing an animal or **human** to induce protection against CDV by administering to the animal or **human** an immunogenic composition comprising a **recombinantly** produced CDV;
(5) a nucleic acid molecule comprising a sequence encoding a genome or antigenome of a CDV, or one or more proteins of a CDV;
(6) a plasmid comprising a **polynucleotide** sequence encoding a genome or antigenome of a CDV, or one or more proteins of CDV;
(7) a host cell transformed with at least one plasmid of (6); and
(8) a nucleotide sequence comprising the sequence of a cDNA clone of a **recombinant** CDV.
ACTIVITY - Antiviral; immunostimulant.
MECHANISM OF ACTION - Gene therapy; vaccine. No supporting data is given.
USE - The **recombinant** virus are useful as vectors for expressing foreign genetic information, e.g. foreign genes, for applications including immunogenic or pharmaceutical compositions for pathogens other than canine distemper, gene therapy, and cell targeting. The **recombinant** CDVs are also used in generating antibodies, prophylactic and therapeutic applications, specifically in treating or ameliorating canine distemper infection, and in preparing **mutant** virus and immunogenic compositions. Protein and nucleotide sequences may be used to design screening systems for compounds that interfere or disrupt normal virus development, via encapsidation, replication, or amplification.
Dwg.0/10

L24 ANSWER 13 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2002-090518 [12] WPIDS
DOC. NO. CPI: C2002-027998
TITLE: An isolated infectious **recombinant** **respiratory syncytial virus** (**RSV**) having one or more shifted **RSV** gene(s) or genome segment(s) within the **recombinant** genome or antigenome, useful as an attenuated vaccine against **RSV** strains.

DERWENT CLASS: B04 D16
INVENTOR(S): BUCHHOLZ, U; COLLINS, P L; KREMPL, C D; MURPHY, B R; WHITEHEAD, S S
PATENT ASSIGNEE(S): (USGO) US GOVERNMENT; (BUCH-I) BUCHHOLZ U; (COLL-I) COLLINS P L; (KREM-I) KREMPL C D; (MURP-I) MURPHY B R; (WHIT-I) WHITEHEAD S S
COUNTRY COUNT: 96
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002000693	A2	20020103	(200212)*	EN	168
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2001068709	A	20020108	(200235)		
US 2002146433	A1	20021010	(200269)		
EP 1294858	A2	20030326	(200323)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002000693	A2	WO 2001-US20107	20010622
AU 2001068709	A	AU 2001-68709	20010622
US 2002146433	A1 Provisional	US 2000-213708P	20000623
		US 2001-887469	20010622
EP 1294858	A2	EP 2001-946696	20010622
		WO 2001-US20107	20010622

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001068709	A Based on	WO 200200693
EP 1294858	A2 Based on	WO 200200693

PRIORITY APPLN. INFO: US 2000-213708P 20000623; US 2001-887469 20010622
AN 2002-090518 [12] WPIDS
AB WO 200200693 A UPAB: 20020221
NOVELTY - An isolated infectious **recombinant respiratory syncytial virus** (**RSV**) having one or more shifted **RSV** gene(s) or genome segment(s) within the **recombinant** genome or antigenome that is/are positionally shifted to a more promoter-proximal or promoter-distal position relative to a position of the **RSV** gene(s) or genome segment(s) within a wild type **RSV** genome or antigenome, is new.
DETAILED DESCRIPTION - An isolated infectious **recombinant respiratory syncytial virus** (**RSV**) comprising a major nucleocapsid (N) protein, a nucleocapsid phosphoprotein (P), a large polymerase protein (L), a RNA polymerase elongation factor, and a partial or complete **recombinant RSV** genome or antigenome having one or more shifted **RSV** gene(s) or genome segment(s) within the **recombinant** genome or antigenome that

is/are positionally shifted to a more promoter-proximal or promoter-distal position relative to a position of the **RSV** gene(s) or genome segment(s) within a wild type **RSV** genome or antigenome.

INDEPENDENT CLAIMS are included for the following:

(1) a method (M1) for stimulating the immune system of an individual to induce protection against **RSV** which comprises administering to the individual an immunologically sufficient amount of the **recombinant RSV** combined with a physiologically acceptable carrier;

(2) an isolated **polynucleotide** molecule comprising a **recombinant RSV** genome or antigenome having one or more shifted **RSV** gene(s) or genome segment(s) within the **recombinant** genome or antigenome that is/are positionally shifted to a more promoter-proximal or promoter-distal position relative to a position of the **RSV** gene(s) or genome segment(s) within a wild type **RSV** genome or antigenome;

(3) a method (M2) for producing an infectious attenuated **recombinant RSV** particle from one or more isolated **polynucleotide** molecules encoding the **RSV**, comprising expressing in a cell or cell-free lysate an expression vector comprising an isolated **polynucleotide** comprising a **recombinant RSV** genome or antigenome having one or more shifted **RSV** gene(s) or genome segment(s) within the **recombinant** genome or antigenome that is/are positionally shifted to a more promoter-proximal or promoter-5 distal position relative to a position of the **RSV** gene(s) or genome segment(s) within a wild type **RSV** genome or antigenome, and **RSV** N, P, L and RNA polymerase elongation factor proteins;

(4) an isolated infectious chimeric **RSV** comprising a major nucleocapsid (N) protein, a nucleocapsid phosphoprotein (P), a large polymerase protein (L), a RNA polymerase elongation factor, and a partial or complete bovine **RSV** background genome or antigenome combined with heterologous gene(s) and/or genome segment(s) of a **human RSV** selected from heterologous gene(s) and/or genome segment(s) of **RSV** NS1, NS2, M, SH, G, and/or F, to form a **human**-bovine chimeric **RSV** genome or antigenome; and

(5) an isolated **polynucleotide** molecule comprising a **recombinant RSV** genome or antigenome comprising a partial or complete bovine **RSV** background genome or antigenome combined with a plurality of heterologous gene(s) and/or genome segment(s) of a **human RSV** selected from heterologous gene(s) and/or genome segment(s) of **RSV** NS1, NS2, M, SH, G, and/or F genes, to form a **human**-bovine chimeric **RSV** genome or antigenome.

ACTIVITY - Antiviral.

No biological data given.

MECHANISM OF ACTION - The **recombinant RSV** elicits an immune response against either **human RSV** A or **RSV** B or both **human RSV** A and **RSV** B (claimed); gene therapy; vaccine.

No biological data given.

USE - The **recombinant RSV** is useful in an attenuated vaccine to elicits an immune response against one or more strains of **RSV**.

Dwg.0/15

L24 ANSWER 14 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2002-620673 [67] WPIDS
CROSS REFERENCE: 1999-621834 [54]; 2002-629646 [68]; 2002-637831 [69]
DOC. NO. CPI: C2002-175516

TITLE: Novel Neospora caninum SAG1 protein useful for producing vaccines against neosporosis and as diagnostic reagents.
DERWENT CLASS: B04 C06 D16
INVENTOR(S): BRAKE, D A; DURTSCHI, B A; KRISHNAN, B R; MADURA, R A; YODER, S C
PATENT ASSIGNEE(S): (PFIZ) PFIZER PROD INC
COUNTRY COUNT: 18
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
EP 1221486	A2	20020710	(200267)*	EN	54

R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU NL PT SE

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
EP 1221486	A2 Div ex	EP 1999-301746	19990309
		EP 2002-2960	19990309

FILING DETAILS:

PATENT NO	KIND	PATENT NO
EP 1221486	A2 Div ex	EP 953641

PRIORITY APPLN. INFO: US 1998-112282P 19981215; US 1998-79389P 19980326
AN 2002-620673 [67] WPIDS
CR 1999-621834 [54]; 2002-629646 [68]; 2002-637831 [69]
AB EP 1221486 A UPAB: 20021031
NOVELTY - A purified or isolated polypeptide (I) chosen from Neospora caninum SAG1 protein (I), a polypeptide having an amino acid sequence that is homologous to an N. caninum SAG1 protein, a polypeptide consisting of a portion of N. caninum SAG1 protein, or polypeptide which is homologous to it, an analog or derivative of (I), and a fusion protein comprising (I), is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated **polynucleotide** molecule (II) comprising a nucleotide sequence encoding a Neospora SAG1 protein, the nucleotide sequence comprising the open reading frame (ORF) of 1263 base pairs (S1), given in the specification from nucleotide 130-1089, or the nucleotide sequence of the SAG1-encoding ORF of plasmid pRC102 (ATCC 209687);

(2) an isolated **polynucleotide** molecule comprising a nucleotide sequence that is homologous to (II);

(3) an isolated **polynucleotide** molecule comprising a nucleotide sequence encoding a polypeptide that is homologous to a polypeptide comprising a 319 residue amino acid sequence (S2), given in the specification;

(4) an isolated **polynucleotide** molecule consisting of a nucleotide sequence that is a substantial portion of any of the above nucleotide sequences;

(5) an isolated **polynucleotide** molecule comprising a nucleotide sequence of 1-129 or 1090-1263 of (S1) or its substantial portion;

(6) an oligonucleotide molecule (III) chosen from (S5);

(7) a **recombinant** vector (IV) comprising a **polynucleotide** molecule comprising a nucleotide sequence encoding

(I);

(8) a transformed host cell comprising (IV);

(9) an isolated antibody (V) that specifically reacts to a N. caninum protein SAG1;

(10) a genetic construct (VI) comprising a **polynucleotide** molecule that can be used to disable a Neospora gene, comprising a **polynucleotide** molecule having a nucleotide sequence that is otherwise the same as a nucleotide sequence encoding a SAG1 protein from N. caninum, or a substantial portion of the nucleotide sequence, but which nucleotide further comprises one or more disabling mutation, or a **polynucleotide** molecule comprising a nucleotide sequence that naturally flanks in situ the ORF of a Neospora SAG1 gene, so that transformation of a Neospora cell with the genetic construct results in disabling of the SAG1 gene;

(11) a Neospora cell (VII) that has been modified by transformation with (VI) so that the SAG1 gene has been disabled;

(12) a vaccine (VIII) against neosporosis, comprising (I), a **polynucleotide** molecule comprising a nucleotide sequence encoding (I) or (VII); and

(13) a kit for vaccinating a mammal against neosporosis comprising a container comprising the above vaccine.

(S5) is aattaaccctcactaaaggg, gtaatacgactcactatagggc, gccgcgacttctttttctct, ctcgatcgcctcctttactg, tgctagtactggcgagtgaa, caggtttgccacacattttt, atgtttcctcctcgggcagtg, tcacgcgacgccagccgctatcg, gccctgacaattcgaccgcc, cccacaacatccaagtcgttc, gttttgcaccatccttagtg, gagagtttgctttgcaccg, and ccagccgagttcgtgttcaga, or aaagctcttcggcagttcaa, ccgcgctaccactttcca, gtaatacgactcactata, catcagagaaactggagt, ggccaagcttgctagtactggcga, and atccaatgcatcttgctgaatgccttaaaag.

ACTIVITY - Protozoacide; Virucide; Antibacterial; Antifungal.

No biological data is given.

MECHANISM OF ACTION - Vaccine.

USE - (I), a **polynucleotide** molecule encoding (I), or (VII) is useful for preparing a vaccine against neosporosis. (VI) is useful for preparing modified Neospora cells, that express a **mutant** phenotype of SAG1. (VIII) is useful for vaccinating a mammal against neosporosis. The second component in the vaccine is capable of inducing, or contributing to the induction of a protective response against a pathogen such as bovine herpes virus, bovine **respiratory syncytial virus**, bovine viral diarrhea virus, **parainfluenza** virus types I, II or III, Leptospira spp., Campylobacter spp., Staphylococcus aureus, Streptococcus agalactiae, Mycoplasma spp., Klebsiella spp., Salmonella spp., rotavirus, coronavirus, rabies, Pasteurella hemolytica, Pasteurella multocida, Clostridia spp., Tetanus toxoid, Escherichia coli, Cryptosporidium spp., Eimeria spp. or Trichomonas spp.. (All claimed). (I) is useful as diagnostic reagents, to screen for Neospora-specific antibodies in blood or serum samples from animals, or as antigens to raise polyclonal or monoclonal antibodies which are useful as diagnostic reagents and to screen for Neospora-specific proteins in cell, tissue or fluid samples from an animal. (III) is useful as primers in amplification of Neospora-specific **polynucleotide** molecule for use, e.g. in differential disease diagnosis, or to encode or act as antisense molecules useful in gene regulation. The sequences of the **polynucleotide** molecules can also be used to design primers for use in isolating homologous genes from other species or strains of Neospora.

Dwg.0/0

L24 ANSWER 15 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2002-637831 [69] WPIDS
CROSS REFERENCE: 1999-621834 [54]; 2002-620673 [67]; 2002-629646 [68]

DOC. NO. CPI: C2002-180167
TITLE: Novel Neospora caninum GRA2 protein useful for producing vaccines against neosporosis and as diagnostic reagents.
DERWENT CLASS: B04 C06 D16
INVENTOR(S): BRAKE, D A; DURTSCHI, B A; KRISHNAN, B R; MADURA, R A; YODER, S C
PATENT ASSIGNEE(S): (PFIZ) PFIZER PROD INC
COUNTRY COUNT: 18
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
EP 1221485	A2	20020710	(200269)*	EN	55

R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU NL PT SE

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
EP 1221485	A2	Div ex	EP 1999-301746	19990309
			EP 2002-2959	19990309

FILING DETAILS:

PATENT NO	KIND		PATENT NO
EP 1221485	A2	Div ex	EP 953641

PRIORITY APPLN. INFO: US 1998-112282P 19981215; US 1998-79389P 19980326
AN 2002-637831 [69] WPIDS
CR 1999-621834 [54]; 2002-620673 [67]; 2002-629646 [68]
AB EP 1221485 A UPAB: 20021026
NOVELTY - A substantially purified or isolated polypeptide (I) chosen from Neospora caninum GRA2 protein (I), a polypeptide with an amino acid sequence that is homologous to an N.caninum GRA2 protein, a polypeptide consisting of a portion of N.caninum GRA2 protein, or polypeptide which is homologous to it, an analog or derivative of (I), and a fusion protein comprising (I), is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:

(1) an isolated **polynucleotide** molecule (II) comprising a nucleotide sequence encoding a Neospora GRA2 protein, the nucleotide sequence comprising the open reading frame (ORF) of 1031 bp (S1) given in the specification from nucleotide 25-660 or the nucleotide sequence of the GRA2-encoding ORF of plasmid pRC5 (ATCC 209686);

(2) an isolated **polynucleotide** molecule comprising a nucleotide sequence that is homologous to (II);

(3) an isolated **polynucleotide** molecule comprising a nucleotide sequence encoding a polypeptide that is homologous to a polypeptide comprising a sequence (S2) of 211 amino acids given in the specification;

(4) an isolated **polynucleotide** molecule consisting of a nucleotide sequence that is a substantial portion of any of the above nucleotide sequences;

(5) an isolated **polynucleotide** molecule comprising a nucleotide sequence of 1-24 or 661-1031 of (S1) or its substantial portion;

(6) an oligonucleotide molecule (III) chosen from (i); (ii); (iii); (iv); (v); (vi); (vii); (viii); (ix); (x); (xi); (xii); (xiii); (xiv); (xv); (xvi); (xvii); (xviii); and (xix), or their complements;

(7) a **recombinant** vector (IV) comprising a **polynucleotide** molecule comprising a nucleotide sequence encoding (I);

(8) a transformed host cell comprising (IV);

(9) an isolated antibody (V) that specifically reacts to a N.caninum protein GRA2;

(10) a genetic construct (VI) comprising a **polynucleotide** molecule that can be used to disable a Neospora gene, comprising a **polynucleotide** molecule having a nucleotide sequence that is otherwise the same as a nucleotide sequence encoding a GRA2 protein from N.caninum, or a substantial portion of the nucleotide sequence, but which nucleotide further comprises one or more disabling mutations, or a **polynucleotide** molecule comprising a nucleotide sequence that naturally flanks in situ the ORF of a Neospora GRA2 gene, such that transformation of a Neospora cell with the genetic construct results in disabling of the GRA2 gene;

(11) a Neospora cell (VII) that has been modified by transformation with (VI) such that the GRA2 gene has been disabled;

(12) a vaccine (VIII) against neosporosis, comprising (I), a **polynucleotide** molecule comprising a nucleotide sequence encoding (I) or (VII); and

(13) a kit for vaccinating a mammal against neosporosis comprising a container comprising (VIII).

aattaaccctcactaaaggg (i);
gtaatacgactcactatagggc (ii);
gccgcgacttctttttctct (iii);
ctcgatcgcctcctttactg (iv);
tgctagtactggcgagtgaa (v);
caggtttgccacacattttt (vi);
atgtttcctcctcgggcagtg (vii);
tcacgcgacgccagccgctatcg (viii);
gccctgacaattcgaccgcc (ix);
cccacaacatccaagtcgttc (x);
gttttgcaccatccttagtg (xi);
gagagtttgctttgcaccg (xii); and
ccagccgagttcgtgttcaga (xiii); or
aaagctcttcggcagttcaa (xiv);
ccgcgctaccactttcca (xv);
gtaatacgactcactata (xvi);
catcagagaaactggagt (xvii);
ggccaagcttgctagtactggcga (xviii); and
atccaatgcatcttgctgaatgccttaaaag (xix).

ACTIVITY - Protozoacide; Virucide; Antibacterial.

MECHANISM OF ACTION - Vaccine.

No suitable data given.

USE - (I), a polynucleotide molecule encoding (I), or (VII) is useful for preparing a vaccine against neosporosis. (VI) is useful for preparing modified Neospora cells, that express a mutant phenotype of GRA2. (VIII) is useful for vaccinating a mammal against neosporosis. The second component in the vaccine is capable of inducing, or contributing to the induction of a protective response against a pathogen such as bovine herpes virus, bovine respiratory syncytial virus, bovine viral diarrhea virus, parainfluenza virus types I, II or III, Leptospira spp., Campylobacter spp., Staphylococcus aureus, Streptococcus agalactiae, Mycoplasma spp., Klebsiella spp., Salmonella spp., rotavirus, coronavirus, rabies, Pasteurella hemolytica, Pasteurella multocida, Clostridia spp., Tetanus toxoid, Escherichia coli, Cryptosporidium spp., Eimeria spp. or Trichomonas spp. (claimed). (I) is useful as diagnostic reagents, to screen for Neospora-specific antibodies in blood or serum samples from animals, or as antigens to raise polyclonal or monoclonal antibodies which

are useful as diagnostic reagents and to screen for Neospora-specific proteins in cell, tissue or fluid samples from an animal. (III) is useful as primers in amplification of Neospora-specific polynucleotide molecule for use, e.g. in differential disease diagnosis, or to encode or act as antisense molecules useful in gene regulation. The sequences of the polynucleotide molecules can also be used to design primers for use in isolating homologous genes from other species or strains of Neospora. Dwg.0/0

L24 ANSWER 16 OF 67 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:430846 BIOSIS
DOCUMENT NUMBER: PREV200100430846
TITLE: Product of infectious **respiratory syncytial virus** from cloned nucleotide sequences.
AUTHOR(S): Collins, Peter L. (1)
CORPORATE SOURCE: (1) Rockville, MD USA
ASSIGNEE: The United States of America as represented by the Department of Health and Human Services
PATENT INFORMATION: US 6264957 July 24, 2001
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (July 24, 2001) Vol. 1248, No. 4, pp. No Pagination. e-file.
ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
AB Isolated **polynucleotide** molecules provide **RSV** genome and antigenomes, including that of **human**, bovine or murine **RSV** or **RSV**-like viruses, and chimera thereof. The **recombinant** genome or antigenome can be expressed with a nucleocapsid (N) protein, a nucleocapsid phosphoprotein (P), a large (L) polymerase protein, and an RNA polymerase elongation factor to produce isolated infectious **RSV** particles. The **recombinant** **RSV** genome and antigenome can be modified to produce desired phenotypic changes, such as attenuated viruses for vaccine use.

L24 ANSWER 17 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2002-147575 [19] WPIDS
DOC. NO. CPI: C2002-045728
TITLE: New synthetic polypeptides having several different segments of at least one parent polypeptide linked together differently compared to the linkage in the parent polypeptide, for inducing immune response against a pathogen or cancer.
DERWENT CLASS: B04 D16
INVENTOR(S): RAMSHAW, I A; THOMSON, S A
PATENT ASSIGNEE(S): (AUSU) UNIV AUSTRALIAN NAT
COUNTRY COUNT: 97
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001090197	A1	20011129	(200219)*	EN	364

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2001059957 A 20011203 (200221)

EP 1285004 A1 20030226 (200319) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001090197	A1	WO 2001-AU622	20010525
AU 2001059957	A	AU 2001-59957	20010525
EP 1285004	A1	EP 2001-933479	20010525
		WO 2001-AU622	20010525

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2001059957	A	Based on	WO 200190197
EP 1285004	A1	Based on	WO 200190197

PRIORITY APPLN. INFO: AU 2000-7761 20000526
AN 2002-147575 [19] WPIDS
AB WO 200190197 A UPAB: 20020321
NOVELTY - A new synthetic polypeptide (I) comprising several different segments of at least one parent polypeptide linked together in a different relationship relative to their linkage in the parent polypeptide to impede, abrogate or otherwise alter at least one function associated with the parent polypeptide.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a synthetic **polynucleotide** encoding (I);

(2) a method for producing the synthetic **polynucleotide** encoding (I);

(3) a synthetic construct comprising a synthetic **polynucleotide** encoding (I);

(4) an immunopotentiating composition, comprising (I), the synthetic **polynucleotide**, or the synthetic construct, and a carrier;

(5) a method for modulating an immune response directed against a pathogen or a cancer, by administering to the patient an immunopotentiating agent selected from (I), the synthetic **polynucleotide**, the synthetic construct, or the composition of (4);

(6) a method for the treatment and/or prophylaxis of a disease or condition, by administering to the patient an immunopotentiating agent selected from (I), the synthetic **polynucleotide**, the synthetic construct, or the composition of (4);

(7) a computer program product for designing the sequence of a synthetic polypeptide comprising:

(a) code that receives as input the sequence of at least one parent polypeptide;

(b) code that fragments the sequence of a respective parent polypeptide into fragments;

(c) code that links together the fragments in a different relationship relative to their linkage in the parent polypeptide sequence; and

(d) a computer readable medium that stores the codes.

(8) a computer for designing the sequence of a synthetic polypeptide comprising:

(a) a machine-readable data storage medium comprising a data storage material encoded with machine-readable data having at least one parent

polypeptide;
(b) a working memory for storing instructions for processing the machine-readable data;
(c) a central-processing unit coupled to the working memory and to the machine readable data storage medium, for processing the machine readable data to provide the synthetic polypeptide sequence; and
(d) an output hardware coupled to the central processing unit, for receiving the synthetic polypeptide sequence.
ACTIVITY - Cytostatic; virucide; antibacterial; antiparasitic; immunostimulant.
MECHANISM OF ACTION - Vaccine.
USE - The synthetic polypeptide is useful for modulating immune responses preferably directed against a pathogen or a cancer, (claimed), (e.g., cancers of the lung, breast, ovary, cervix, colon, head and neck, pancreas, prostate, stomach, bladder, kidney, bone liver, esophagus, brain, testicle, uterus), as potentiating agents. Compositions comprising the polypeptide may be used in the treatment or prophylaxis against viral, (claimed), (such as infections caused by HIV, hepatitis, influenza, Japanese encephalitis virus, Epstein-Barr virus and **respiratory syncytial virus**), bacterial (e.g., infections caused by Neisseria, Meningococcal, Haemophilus, Salmonella, Streptococcal, Legionella and Mycobacterium or parasitic (e.g., infections caused by Plasmodium, Schistosoma, Leishmania, Trypanosoma, Toxoplasma and Giardia) infections.
Dwg.0/30

L24 ANSWER 18 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2002-066532 [09] WPIDS
DOC. NO. NON-CPI: N2002-049388
DOC. NO. CPI: C2002-019839
TITLE: New Pellino polypeptides for identifying compounds that alter polypeptide activity, treating pathogenic infection or inhibiting apoptosis, are capable of stimulating nuclear factor-kappaB- or p38-dependent transcription.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): BIRD, T A; COSMAN, D J
PATENT ASSIGNEE(S): (IMMV) IMMUNEX CORP; (BIRD-I) BIRD T A; (COSM-I) COSMAN D J
COUNTRY COUNT: 95
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001083739	A2	20011108	(200209)*	EN	70
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2001059217	A	20011112	(200222)		
US 2002168683	A1	20021114	(200277)		
EP 1290160	A2	20030312	(200320)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE

WO 2001083739 A2	WO 2001-US13676	20010427
AU 2001059217 A	AU 2001-59217	20010427
US 2002168683 A1 Provisional	US 2000-200198P	20000428
	US 2001-843905	20010427
EP 1290160 A2	EP 2001-932711	20010427
	WO 2001-US13676	20010427

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2001059217	A	Based on	WO 200183739
EP 1290160	A2	Based on	WO 200183739

PRIORITY APPLN. INFO: US 2000-200198P 20000428; US 2001-843905 20010427

AN 2002-066532 [09] WPIDS

AB WO 200183739 A UPAB: 20020208

NOVELTY - An isolated polypeptide capable of stimulating nuclear factor (NF)-kappa(k)B-dependent transcription or p38-dependent transcription, referred as Pellino polypeptide (I), comprising a sequence of 418 (S1), 420 (S2) or 445 (S3) amino acids defined in the specification, an allelic **variant**, fragment of 20 contiguous amino acids of (I), is new.

DETAILED DESCRIPTION - An isolated polypeptide capable of stimulating nuclear factor (NF)-kappa(k)B-dependent transcription or p38-dependent transcription, referred as Pellino polypeptide (I), comprising:

(A) a fully defined sequence of 418 (S1), 420 (S2) or 445(S3) amino acids, as given in the specification;

(B) amino acid sequence chosen from:

(a) amino acids 130-134 to 187-191, or 1-10 to 409-418 of (S1);

(b) 132-136 to 189-193, or 1-10 to 410-419 of (S2); and

(c) 155-160 to 212-217; 1-10 to 435-445 of (S3);

(C) an allelic **variant** of (I);

(D) fragment comprising 20 contiguous amino acids and capable of stimulating NF-kB-dependent transcription or comprising RING-finger-like domain amino acid sequences;

(E) an amino acid sequence comprising at least 20 amino acids and sharing 85%, 95%, 97.5%, 99% and 99.5% identity with (S1)-(S3) capable of stimulating NF-kB-dependent transcription; and

(F) an amino acid sequence of (e) which binds to an antibody that also binds to a polypeptide comprising an amino acid sequence of any of (a)-(d), capable of stimulating NF-kB-dependent transcription.

INDEPENDENT CLAIMS are also included for the following:

(1) an isolated dominant-negative Pellino polypeptide (II) capable of inhibiting NF-kB-dependent transcription or p38-dependent transcription, comprising:

(i) a sequence of (S1), (S2) or (S3), where amino acids 1-x1 have been deleted from the sequence, where x1 is amino acids 50-98;

(ii) sequence (S1), where amino acids 99-178 to 100-179 have been deleted, sequence (S2), where amino acids 1-180 to 2-181 have been deleted, or sequence (S3), where amino acids 1-206 to 2-207 have been deleted;

(iii) an amino acid sequence (S1), (S2) or (S3), where one or more cysteine residues of the RING-finger-like domain have been deleted or replaced by non-cysteine residues;

(iv) allelic **variant** of (VI); and

(v) fragments of the above sequences comprising RING-finger-like domain amino acid sequences and capable of inhibiting NF-kB-dependent transcription;

(2) an isolated nucleic acid (III) encoding (I) or (II);

(3) an expression vector (IV) comprising (III);

(4) a **recombinant** host cell (V) comprising at least one **recombinant** nucleic acid comprising (III);

(5) producing (I) or (II);

(6) a polypeptide produced by the above method;

(7) an isolated antibody (VI) that binds to (I) or (II) and inhibits the activity of the polypeptide;

(8) an isolated genomic nucleic acid corresponding to the nucleic acid encoding (I);

(9) designing an inhibitor of (I), by determining the three-dimensional structure of such polypeptide, analyzing the structure for the likely binding sites of substrates, synthesizing a molecule that incorporates a predicted reactive site, and determining the polypeptide-inhibiting activity of the molecule;

(10) stimulating NF-kB-dependent transcription, by providing (I) or agonists of (I);

(11) inhibiting NF-kB-dependent transcription, by providing an antagonist of (I), or (II) or agonists of (II);

(12) an isolated polypeptide (VII) comprising

(i) a sequence of (S1), (S2) or (S3), where amino acids 1-x1 have been deleted from the sequence, where x1 is amino acids 50-98;

(ii) a sequence (S1), where amino acids 99-157 to 100-158 have been deleted, sequence (S2), where amino acids 1-159 to 2-160 have been deleted; or

(iii) sequence (S3), where amino acids 1-184 to 2-185 have been deleted; and

(13) an isolated nucleic acid encoding (VII).

ACTIVITY - Virucide; Antibacterial; Fungicide; Protozoacide; Antiasthmatic; Antirheumatic; Antiarthritic; Antiulcer; Antiinflammatory; Antiatherosclerotic; Neuroprotective; Nootropic. No biological data provided.

MECHANISM OF ACTION - Modulator of NF-kappaB-dependent transcription or p38-dependent transcription. No biological data is given.

USE - (I) and (II) are useful for identifying compounds that alter the Pellino polypeptide and Pellino dominant-negative activity, respectively. The method can also be carried out using cells expressing (I) or (II). (claimed). Pellino polypeptides are also useful for identifying compounds that inhibit the binding activity of the polypeptides and to study cell-signal transduction. (I) or (II) is useful for preventing or treating infection by a pathogen such as virus, bacterial, fungi, algae or protozoa, or inhibiting apoptosis. Dominant-negative Pellino polypeptides are useful for treating inflammatory conditions such as asthma, rheumatoid arthritis, inflammatory bowel disease, Crohn's disease, ulcerative colitis, atherosclerosis and Alzheimer's disease, and also for inhibiting mitogen activated protein (MAP) kinase-activated pathways. (III) is useful as probe or primer to identify DNA encoding proteins having Pellino activity, to construct expression vectors and develop transgenic and knockout cells and animals and to screen cDNA libraries derived from other mammalian species. Pellino polypeptides and polynucleotides are useful to identify small molecule inhibitors of protein association or function of Pellino, and other molecules involved in IL-1 signaling. Pellino polypeptides and its fragments are useful to generate antibodies which are useful to purify the polypeptides and to detect the presence of the polypeptides in vitro or in vivo.

Dwg.0/0

L24 ANSWER 19 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2001-502717 [55] WPIDS
DOC. NO. CPI: C2001-151257

TITLE: A **recombinant** adeno-associated virus vector whose genome comprises a **polynucleotide** sequence(s) encoding an antibody useful for the treatment of viral infection, asthma, allergy and macular degeneration.

DERWENT CLASS: B04 D16

INVENTOR(S): KOENIG, S

PATENT ASSIGNEE(S): (MEDI-N) MED IMMUNE INC; (KOEN-I) KOENIG S; (MEDI-N) MEDIMMUNE INC

COUNTRY COUNT: 94

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001059142	A1	20010816	(200155)*	EN	47

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW

US 2001034062 A1 20011025 (200170)

AU 2001038079 A 20010820 (200175)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001059142	A1	WO 2001-US4150	20010209
US 2001034062	A1 Provisional	US 2000-182312P	20000209
		US 2001-781052	20010209
AU 2001038079	A	AU 2001-38079	20010209

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001038079	A Based on	WO 200159142

PRIORITY APPLN. INFO: US 2000-182312P 20000209; US 2001-781052 20010209

AN 2001-502717 [55] WPIDS

AB WO 200159142 A UPAB: 20010927

NOVELTY - A **recombinant** adeno-associated virus (rAAV) vector (I) whose genome comprises a **polynucleotide** sequence(s) (II) encoding a polypeptide(s) (III) of an antibody, is new. The **polynucleotide** is operably linked to control elements that direct intracellular transcription and translation of the **polynucleotide** when (I) is inserted into a mammalian cell.

DETAILED DESCRIPTION - An INDEPENDENT CLAIMS is also included for a **recombinant** cell (IV) whose genome comprises (I).

ACTIVITY - Antibacterial; antifungal; antiviral; antiparasitic; antiasthmatic; antiallergic; opthalmic; antiinflammatory.

MECHANISM OF ACTION - IgG.

No supporting data is given.

USE - (I) is useful for treating viral infection, asthma, allergy and macular degeneration when âdministered into tissues of a patient especially respiratory tissue, muscle tissue or opthalmic tissue. (IV) is useful for treating respiratory disease especially bronchitis, bronchiolitis or pneumonia (all claimed).

ADVANTAGE - The vector provides a continuous measured amount of antibody at a relatively low cost for the treatment of infections eliminating the need for large doses of antibody to be given intravenously.
Dwg.0/0

L24 ANSWER 20 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2001-483242 [52] WPIDS
CROSS REFERENCE: 2001-483240 [52]
DOC. NO. CPI: C2001-144933
TITLE: Identifying oligonucleotides with transcriptional/translational regulatory activity in eukaryotic cells by integrating an oligonucleotide into cell genome and detecting a change in expression of expressible **polynucleotides**.
DERWENT CLASS: B04 D16
INVENTOR(S): CHAPPELL, S A; EDELMAN, G M; JONES, F S; MAURO, V P; MEECH, R; OWENS, G; CHAPPELL, G M
PATENT ASSIGNEE(S): (SCRI) SCRIPPS RES INST
COUNTRY COUNT: 95
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001055371	A1	20010802	(200152)*	EN	172
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2001031206	A	20010807	(200174)		
EP 1259602	A1	20021127	(200302)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001055371	A1	WO 2001-US2733	20010126
AU 2001031206	A	AU 2001-31206	20010126
EP 1259602	A1	EP 2001-903383	20010126
		WO 2001-US2733	20010126

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2001031206	A	Based on	WO 200155371
EP 1259602	A1	Based on	WO 200155371

PRIORITY APPLN. INFO: US 2001-261312P 20010112; US 2000-178816P 20000128; US 2000-186496P 20000302; US 2000-207804P 20000530; US 2000-230852P 20000907; US 2000-230956P 20000907
AN 2001-483242 [52] WPIDS
CR 2001-483240 [52]
AB WO 200155371 A UPAB: 20030111
NOVELTY - Identifying (M) oligonucleotide (I) with

transcriptional/translational regulatory activity in eukaryotic cell (EC) by integrating (I) into genome of ECs so that it is linked to an expressible **polynucleotide** (II) or contacting EC with library of vectors obtained by cloning a library of (I) into multiple copies of expression vectors comprising (II), and detecting changes in expression of (II).

DETAILED DESCRIPTION - Identifying (M) oligonucleotide (I) with transcriptional/translational regulatory activity in eukaryotic cell (EC) by integrating (I) into genome of ECs so that it is linked to an expressible **polynucleotide** (II) or contacting EC with library of vectors obtained by cloning a library of (I) into multiple copies of expression vectors comprising (II), and detecting changes in expression of (II).

(M) involves:

(a) integrating (I) to be examined for the regulatory activity into genome of EC, where (I) is operatively linked to an expressible **polynucleotide** (II), and detecting a change in the level of expression of (II) in the presence of (I) as compared to absence of (I), therefore identifying (I) as having transcriptional regulatory activity in EC; or

(b) cloning a library of (I) to be examined for transcriptional or translational regulatory activity into multiple copies of an expression vector comprising (II), where (I) is operatively linked to (II) to obtain a library of vector, contacting the library of vectors with EC under conditions so that the vectors are introduced into EC and integrated into a chromosome in EC, and detecting expression of (II) operatively linked to (I) at a level other than level of expression of (II) in the absence of (I), therefore identifying (I) as having transcriptional regulatory activity in EC.

INDEPENDENT CLAIMS are also included for the following:

(1) an isolated transcriptional or translational regulatory element (III) obtained by M1;

(2) a **recombinant** nucleic acid molecule (IV) comprising a number of operatively linked (III);

(3) an integrating expression vector (V) comprising in operative linkage in a 5' to 3' orientation, a long terminal repeat (LTR) containing a immediate early gene promoter, an R region, a U5 region, a truncated gag gene comprising sequences required for retrovirus packaging, a di-cistronic reporter cassette comprising a first reporter cassette, a spacer sequence comprising an internal ribosome entry site (IRES) or a cloning site, a second reporter cassette, and a regulatory cassette comprising a promoter and optionally a cloning site, and an LTR;

(4) a kit (VI) comprising (III) or (VI); and

(5) an isolated transcriptional regulatory element (VII) selected from a sequence comprising 66 or 30 base pairs fully defined in the specification.

USE - (M) Is useful for identifying an oligonucleotide having transcriptional or translational activity in a eukaryotic cell (claimed).

(M) is useful for identifying synthetic transcriptional or translational regulatory elements. (III) is useful in a variety of gene expression configurations for regulating control of expression, and in expression vectors for controlling gene expressions in diagnostic and therapeutic applications.

ADVANTAGE - (M) Quickly and conveniently screens a large number of oligonucleotides to identify those having transcriptional or translational regulatory activity.

Dwg.0/6

L24 ANSWER 21 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2001-356173 [37] WPIDS

CROSS REFERENCE: 1999-045317 [04]; 2001-091890 [10]
DOC. NO. CPI: C2001-110518
TITLE: Isolated infectious chimeric **parainfluenza** virus (PIV), useful in an attenuated vaccine to elicits an immune response against one or more virus(es) selected from **human** PIV1 (HPIV1), HPIV2 and HPIV3.
DERWENT CLASS: B04 D16 P32
INVENTOR(S): COLLINS, P L; DURBIN, A P; MURPHY, B R; SCHMIDT, A C; SKIADOPOULOS, M H; TAO, T
PATENT ASSIGNEE(S): (USSH) US DEPT HEALTH & HUMAN SERVICES; (COLL-I) COLLINS P L; (DURB-I) DURBIN A P; (MURP-I) MURPHY B R; (SCHM-I) SCHMIDT A C; (SKIA-I) SKIADOPOULOS M H; (TAOT-I) TAO T
COUNTRY COUNT: 95
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001042445	A2	20010614	(200137)*	EN	305
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2001020731	A	20010618	(200161)		
EP 1179054	A2	20020213	(200219)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL RO SI					
CN 1347453	A	20020501	(200252)		
US 2002155581	A1	20021024	(200273)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 2001042445	A2		WO 2000-US33293	20001208
AU 2001020731	A		AU 2001-20731	20001208
EP 1179054	A2		EP 2000-984052	20001208
			WO 2000-US33293	20001208
CN 1347453	A		CN 2000-805939	20001208
US 2002155581	A1	Provisional	US 1997-47575P	19970523
		Provisional	US 1997-59385P	19970919
		CIP of	US 1998-83793	19980522
		Provisional	US 1999-170195P	19991210
			US 2000-733692	20001208

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2001020731	A	Based on	WO 200142445
EP 1179054	A2	Based on	WO 200142445

PRIORITY APPLN. INFO: US 1999-459062 19991210; US 1999-170195P 19991210; US 1999-458813 19991210; US 1997-47575P 19970523; US 1997-59385P 19970919; US 1998-83793 19980522; US 2000-733692 20001208
AN 2001-356173 [37] WPIDS
CR 1999-045317 [04]; 2001-091890 [10]

AB WO 200142445 A UPAB: 20021118
NOVELTY - An isolated infectious chimeric **parainfluenza** virus (PIV), is new.

DETAILED DESCRIPTION - An isolated infectious chimeric **parainfluenza** virus (PIV), is new.

The virus comprises a major nucleocapsid protein (N), a nucleocapsid phosphoprotein (P), a large polymerase protein (L), and a partial or complete PIV vector background genome, or antigenome combined with one or more heterologous gene(s) or genome segment(s) encoding one or more antigenic determinants of one or more heterologous pathogen(s) to form a chimeric genome or antigenome.

INDEPENDENT CLAIMS are also included for the following:

(1) a method for stimulating the immune system of an individual to induce protection against PIV, comprising administering an immunologically sufficient amount of the chimeric PIV;

(2) a method for sequential immunization to stimulate the immune system of an individual to induce protection against multiple pathogens comprising administering to a newborn to 4 month old infant an immunologically sufficient amount of a first attenuated chimeric **human** PIV (HPIV) expressing an antigenic determinant of a non-PIV pathogen and one or more antigenic determinants of HPIV3 and subsequently administering an immunologically sufficient amount of a second attenuated chimeric HPIV expressing an antigenic determinant of a non-PIV pathogen and one or more antigenic determinants of HPIV1 or HPIV2;

(3) an isolated **polynucleotide** comprising a chimeric PIV genome or antigenome which includes a partial or complete PIV background genome or antigenome combined with one or more heterologous gene(s) or genome segment(s) encoding one or more antigenic determinant(s) of one or more heterologous pathogen(s) to form a chimeric PIV genome or antigenome;

(4) a method for producing an infectious attenuated chimeric PIV particle from one or more isolated **polynucleotide** molecules encoding the PIV, comprising expressing in a cell or cell-free lysate an expression vector comprising an isolated **polynucleotide** comprising a partial or complete PIV vector genome or antigenome of a **human** or bovine PIV combined with one or more heterologous gene(s) or genome segment(s) encoding one or more antigenic determinant(s) of one or more heterologous pathogen(s) to form a chimeric PIV genome or antigenome, and PIV N, P and L proteins;

(5) an expression vector comprising an operably linked transcriptional promoter, a **polynucleotide** sequence which includes a partial or complete PIV vector genome or antigenome of a **human** or bovine PIV combined with one or more heterologous gene(s) or genome segment(s) encoding one or more antigenic determinant(s) of one or more heterologous pathogen(s) to form a chimeric PIV genome or antigenome, and a transcriptional terminator; and

(6) an isolated infectious **recombinant** PIV comprising a N protein, a P, a L, and a PIV genome or antigenome having a **polynucleotide** insertion of between 150 nucleotides and 4000 nucleotides in length in a non-coding region (NCR) of the genome or antigenome or as a separate gene unit (GU), the **polynucleotide** insertion lacking a complete open reading frame (ORF) and specifying an attenuated phenotype in the **recombinant** PIV.

ACTIVITY - Antiviral.

Chimpanzees in groups of 4 were inoculated intranasally and intratracbeally with 105 TCID50 of rPIV3-2TM or PIV2/V94 on day 0. NT swab specimens (day 1 to 12) and tracheal lavage (days 2, 4, 6, 8, and 10) samples were collected. Virus titer was determined as previously described (Durbin et al., Virology 261:319-30, 1999), and results are expressed as log10 TCID50/ml. rPIV3-2TM had a lower peak titer than its wild type parent PIV2/V94 and was shed for a significantly shorter duration than

PIV2/94, indicating that rPIV3-2TM is attenuated in chimpanzees. PIV2/94 wild-type virus replicates to low levels in chimpanzees compared to hamsters and AFGs (undefined), while rPIV3-2TM virus was attenuated in each of these model hosts.

MECHANISM OF ACTION - Anti-PIV vaccine.

USE - The chimeric PIV is useful in an attenuated vaccine to elicits an immune response against one or more virus(es) selected from HPIV1, HPIV2 and HPIV3. Preferably, the chimeric PIV elicits an immune response against HPIV3 and another virus selected from HPIV1 or HPIV2. The chimeric PIV may also elicits a polyspecific immune response against HPIV3 and measles or **respiratory syncytial virus**. An immunospecific composition may also contain two chimeric PIVs, where the first chimeric PIV elicits an immune response against HPIV3 and the second chimeric PIV elicits an immune response against HPIV1 or HPIV2, and where both the first and second chimeric PIVs elicit an immune response against the non-PIV pathogen (all claimed).

Dwg.0/21

L24 ANSWER 22 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2001-389720 [41] WPIDS
DOC. NO. CPI: C2001-118754
TITLE: Expression vectors comprising a first, crippled selectable marker and a second, amplifiable selectable marker for improved production of polypeptides.
DERWENT CLASS: B04 D16
INVENTOR(S): INNIS, M; SCOTT, E; SCOTT, E M
PATENT ASSIGNEE(S): (CHIR) CHIRON CORP
COUNTRY COUNT: 88
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001032901	A1	20010510	(200141)*	EN	71
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG UZ VN YU ZA ZW					
AU 2000024005	A	20010514	(200149)		
US 2001024807	A1	20010927	(200173)		27
US 6316253	B1	20011113	(200216)		17
EP 1228237	A1	20020807	(200259)	EN	
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					
US 6451539	B2	20020917	(200264)		
JP 2003513635	W	20030415	(200328)		82

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 2001032901	A1		WO 1999-US31275	19991230
AU 2000024005	A		AU 2000-24005	19991230
US 2001024807	A1	Provisional	US 1999-162930P	19991101
		Div ex	US 1999-475460	19991230
			US 2000-748061	20001222
US 6316253	B1	Provisional	US 1999-162930P	19991101
			US 1999-475460	19991230
EP 1228237	A1		EP 1999-967788	19991230
			WO 1999-US31275	19991230

US 6451539	B2	Provisional	US 1999-162930P	19991101
		Div ex	US 1999-475460	19991230
			US 2000-748061	20001222
JP 2003513635	W		WO 1999-US31275	19991230
			JP 2001-535583	19991230

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000024005	A	Based on	WO 200132901
EP 1228237	A1	Based on	WO 200132901
US 6451539	B2	Div ex	US 6316253
JP 2003513635	W	Based on	WO 200132901

PRIORITY APPLN. INFO: US 1999-162930P 19991101; US 1999-475460 19991230; US 2000-748061 20001222

AN 2001-389720 [41] WPIDS

AB WO 200132901 A UPAB: 20030501

NOVELTY - Expression vector (I) comprising:

(a) a first **polynucleotide** encoding a first, crippled selectable marker;

(b) a second **polynucleotide** encoding a heterologous polypeptide of interest; and

(c) a third **polynucleotide** encoding a second, amplifiable selectable marker, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a plasmid (II) selected from pESN1dhfr, pESN2dhfr, pES3dhfr, pneo1dhfr5'del, pneo3dhfr5'del, pneo2dhfr5'del, and pdhfr3'del;

(2) a method (M1) for producing a polypeptide (III) in a host cell comprising:

(a) introducing (I) or (II) into host cells;

(b) selecting host cells that express the first and second selectable markers under conditions that select for stably integrated expression vectors;

(c) growing the stably-transfected host cells under conditions that favor expression of the polypeptide; and

(d) isolating the polypeptide;

(3) a host cell line (IV) that produces (III) using (M1);

(4) a transfection system (V) comprising:

(a) a first construct (VI) comprising a sequence encoding a first selectable marker and a sequence encoding a second selectable marker, where the second marker contains at least one disabling mutation in its coding sequence; and

(b) a second construct (VII) comprising a **polynucleotide** sequence (transgene) and a sequence encoding a third selectable marker, where the third marker is the same as the second marker except that it contains at least one disabling mutation that is in a different region of the coding sequence that of the second marker;

(5) a method (M2) for producing a mammalian cell line for expression of a **polynucleotide** sequence, comprising:

(a) introducing (VI) into a mammalian cell;

(b) selecting for a mammalian cell expressing the first marker;

(c) introducing (VII); and

(d) selecting for a cell line expressing a functional product encoded by the second marker, where the product is encoded by a sequence produced by a **recombinant** event between the second and third markers, and the resulting cell is capable of expressing the **polynucleotide** sequence;

(6) a mammalian cell line (VIII) produced by (M2)

(7) a method (M3) for producing (III) in a host mammalian cell comprising (M2) and further comprising culturing the cell to produce (III);

(8) a mammalian cell line (IX) that produces (III).

USE - The vectors (I) are useful for producing a polypeptide such as CAB-2, CAB-4, uPAR, VEGF-D (all undefined) and a viral protein, especially a viral glycoprotein (claimed).

ADVANTAGE - The expression vectors solve the problems of low yield and varied expression levels by using a crippled first selectable marker linked to a transgene and a second, amplifiable marker, which contains a disabling mutation.

Cells transformed with the vectors are grown in the presence of the appropriate substrate for the first selectable marker, for example, G418 if the first marker encodes neomycin. Cells that survive selection in high concentration of the antibiotic have integrated the neomycin resistance gene at a high expression locus. Furthermore, homologous **recombination** between the second amplifiable marker from one vector with the same disabled marker from another vector (but in a different region of the coding sequence) generates a functional second marker as well as enabling insertion of the desired gene at a high expression locus.

The tedious process of identification of high expression loci in mammalian cells is eliminated and provides an efficient mechanism by which any desired polypeptide can be expressed at high levels using the novel cell lines generated.

In addition, altering the transgene such that aberrant splicing is corrected may increase expression.

Dwg.0/10

L24 ANSWER 23 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2001-138654 [14] WPIDS
CROSS REFERENCE: 2002-188688 [24]
DOC. NO. CPI: C2001-041027
TITLE: New isolated **polynucleotide** useful for outer membrane vesicle preparation from Gram-negative bacterial strain for vaccination of microbial infections.
DERWENT CLASS: B04 D16
INVENTOR(S): BERTHET, F J; DALEMANS, W L J; DENOEL, P; DEQUESNE, G; FERON, C; LOBET, Y; POOLMAN, J; THIRY, G; THONNARD, J; VOET, P; DALEMANS, W L; LHONNARD, J
PATENT ASSIGNEE(S): (SMIK) SMITHKLINE BEECHAM BIOLOGICALS; (SMIK) SMITHKLINE BEECHAM BIOLOGICALS SA
COUNTRY COUNT: 95
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001009350	A2	20010208	(200114)*	EN	127
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2000068336	A	20010219	(200129)		
NO 2002000506	A	20020402	(200235)		
BR 2000012974	A	20020507	(200238)		
CZ 2002000403	A3	20020515	(200241)		
EP 1208214	A2	20020529	(200243)	EN	

R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI
KR 2002027514 A 20020413 (200267)
HU 2002003056 A2 20021228 (200308)
CN 1377415 A 20021030 (200314)
JP 2003506049 W 20030218 (200315) 189

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001009350	A2	WO 2000-EP7424	20000731
AU 2000068336	A	AU 2000-68336	20000731
NO 2002000506	A	WO 2000-EP7424	20000731
		NO 2002-506	20020131
BR 2000012974	A	BR 2000-12974	20000731
		WO 2000-EP7424	20000731
CZ 2002000403	A3	WO 2000-EP7424	20000731
		CZ 2002-403	20000731
EP 1208214	A2	EP 2000-956369	20000731
		WO 2000-EP7424	20000731
KR 2002027514	A	KR 2002-701441	20020201
HU 2002003056	A2	WO 2000-EP7424	20000731
		HU 2002-3056	20000731
CN 1377415	A	CN 2000-813842	20000731
JP 2003506049	W	WO 2000-EP7424	20000731
		JP 2001-514142	20000731

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000068336	A	Based on	WO 200109350
BR 2000012974	A	Based on	WO 200109350
CZ 2002000403	A3	Based on	WO 200109350
EP 1208214	A2	Based on	WO 200109350
HU 2002003056	A2	Based on	WO 200109350
JP 2003506049	W	Based on	WO 200109350

PRIORITY APPLN. INFO: GB 1999-18319 19990803
AN 2001-138654 [14] WPIDS
CR 2002-188688 [24]
AB WO 200109350 A UPAB: 20030303
NOVELTY - An isolated **polynucleotide** sequence which hybridizes under highly stringent conditions to at least a 30 nucleotide portion of 80 sequences described in the specification.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for:
(1) a genetically-engineered outer membrane vesicle (bleb) preparation from a Gram-negative bacterial strain characterized in that the preparation is obtainable by employing a process comprising:
(a) introducing a heterologous gene, optionally controlled by a strong promoter sequence, into the chromosome by homologous **recombination**; and
(b) making blebs from the strain;
(2) a vaccine comprising a bleb preparation and a pharmaceutically acceptable excipient;
(3) a vector suitable for performing **recombination** events;
(4) a modified Gram-negative bacterial strain from which the bleb preparation is made;
(5) an immuno-protective and non-toxic Gram-negative bleb, ghost, or

killed whole cell vaccine suitable for paediatric use.

ACTIVITY - Antiviral; Antibacterial; Antifungal.

Animals were immunized three times with 5 micro g of the different OMVs absorbed on Al(OH)3 on days 0, 14, and 28. Bleedings were done on days 28 and 35, and they were challenged on day 35. The challenge dose was 20 X LD50 (approx. 10 to the power of 7 CFU/mouse). Mortality rate was monitored for 7 days after challenge.

OMVs injected were:

Group1: Cps-, PorA+
Group2: Cps-, PorA-
Group3: Cps-, PorA-, NspA+
Group4: Cps-, PorA-, Omp85+
Group5: Cps-, PorA-, Hsf+

24 hours after the challenge, there was 100% mortality in the negative control group, while mice immunized with the 5 different OMVs preparations were still alive. Sickness was also monitored during the 7 days and the mice immunized with the NSPA over-expressed blebs appeared to be less sick than the other groups. PorA present in PorA+ blebs is likely to confer extensive protection against infection by the homologous strain. However, protection induced by PorA-up-regulated blebs is likely to be due at least to some extent, to the presence of increased amount of NspA, OMP85 or Hsf.

MECHANISM OF ACTION - Vaccine.

USE - The claimed **polynucleotide** sequence is used in performing a homologous **recombination** event within 1000 base pairs upstream of a Gram-negative bacterial chromosomal gene in order to either increase or decrease expression of the gene. The bleb preparation is useful in the manufacture of a medicament for immunizing a **human** host against a disease caused by infection of one or more of the following: Neisseria meningitidis, Neisseria gonorrhoeae, Haemophilus influenza, Moraxella catarrhalis, Pseudomonas aeruginosa, Chlamydia trachomatis, and Chlamydia pneumonia. The invention is useful for immunizing a **human** host against the diseases caused by the above. The invention also provides immunization against the influenza virus. Immuno-protective and non-toxic Gram-negative bleb, ghost, or killed whole cell vaccines are useful for paediatric use (all claimed).

ADVANTAGE - The vaccine is more immunogenic, less toxic, and safer.

Dwg.0/17

L24 ANSWER 24 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2001-123320 [13] WPIDS
DOC. NO. CPI: C2001-035889
TITLE: Producing a **recombinant** mumps virus (MUV), useful as a mumps vaccine, by transfecting or transforming a host cell with a transcription vector comprising a MUV genome or antigenome, and an expression vector encoding trans-acting proteins.
DERWENT CLASS: B04 D16
INVENTOR(S): CLARKE, D K; JOHNSON, E J; SIDHU, M S; UDEM, S A
PATENT ASSIGNEE(S): (AMHP) AMERICAN HOME PROD CORP; (AMHP) WYETH INC; (AMHP) WYETH
COUNTRY COUNT: 92
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001009309	A2	20010208	(200113)*	EN	133

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES

FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS
LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL
TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2000065149 A 20010219 (200129)
EP 1218499 A2 20020703 (200251) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT
RO SE SI
JP 2003506039 W 20030218 (200315) 135
CN 1384877 A 20021211 (200324)
KR 2002092898 A 20021212 (200328)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001009309	A2	WO 2000-US21192	20000802
AU 2000065149	A	AU 2000-65149	20000802
EP 1218499	A2	EP 2000-952452	20000802
		WO 2000-US21192	20000802
JP 2003506039	W	WO 2000-US21192	20000802
		JP 2001-514101	20000802
CN 1384877	A	CN 2000-813773	20000802
KR 2002092898	A	KR 2002-701457	20020201

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000065149	A	Based on	WO 200109309
EP 1218499	A2	Based on	WO 200109309
JP 2003506039	W	Based on	WO 200109309

PRIORITY APPLN. INFO: US 2000-213654P 20000623; US 1999-146664P 19990802
AN 2001-123320 [13] WPIDS
AB WO 200109309 A UPAB: 20010307
NOVELTY - A new method (M1) for producing a **recombinant** mumps virus (MUV) comprises transfecting or transforming, in a rescue composition media, a host cell with:

(a) a transcription vector comprising a sequence encoding a genome or antigenome of mumps virus and

(b) an expression vector comprising sequences encoding trans-acting proteins (NP, P and L) necessary for encapsidation, transcription and replication.

DETAILED DESCRIPTION - A new method (M1) for producing a **recombinant** mumps virus (MUV) comprises transfecting or transforming, in media of a rescue composition, at least one host cell with:

(a) a transcription vector comprising a nucleic acid which comprises a sequence encoding a genome or antigenome of MUV, or its **variant polynucleotide** sequence; and

(b) at least one expression vector which comprises one more nucleic acid molecule(s) comprising a sequence encoding the trans-acting proteins (NP (nucleocapsid), P and L) necessary for encapsidation, transcription and replication.

The method is carried out under conditions sufficient to permit the co-expression of the vectors and the production of the **recombinant** virus.

INDEPENDENT CLAIMS are also included for the following:

(1) a **recombinant** MUV prepared from M1;

(2) an immunogenic composition (C1) comprising the **recombinant** MUV of (1);
(3) a method for immunizing an individual to induce protection against MUV, comprising administering C1;
(4) a nucleic acid (N1) comprising a sequence encoding a genome or antigenome of MUV;
(5) a nucleic acid (N2) comprising a sequence encoding one or more proteins of the MUV;
(6) a plasmid comprising a **polynucleotide** sequence encoding a genome or antigenome of MUV, or one or more proteins of the MUV;
(7) a host cell transformed with the plasmid of (6); and
(8) a nucleotide sequence comprising the sequence of a cDNA clone of a **recombinant** MUV.
ACTIVITY - Antiviral.
No biological data given.
MECHANISM OF ACTION - Gene therapy; vaccine.
No biological data given.
USE - The **recombinantly** produced MUV are useful in antibody generation, diagnostic, prophylactic and therapeutic applications, cell targeting, gene therapy, **mutant** virus preparation and immunogenic composition preparation.
The method may also produce an attenuated virus for use as a vaccine for preventing or ameliorating mumps infection.
ADVANTAGE - The virus has an ability to induce long-lasting immunity with a single dose and a relatively low level of genome **recombination**.
Dwg.0/15

L24 ANSWER 25 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2001-191424 [19] WPIDS
DOC. NO. CPI: C2001-057330
TITLE: Infectious **respiratory syncytial virus** particle, useful for producing vaccines, comprises a viral genome or antigenome with a deletion in an accessory gene.
DERWENT CLASS: B04 D16
INVENTOR(S): BRYANT, M; JIN, H; LI, S; TANG, R
PATENT ASSIGNEE(S): (AVIR-N) AVIRON
COUNTRY COUNT: 94
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001008703	A1	20010208	(200119)*	EN	127
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW					
AU 2000065118	A	20010219	(200129)		
EP 1204424	A1	20020515	(200239)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SI					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001008703	A1	WO 2000-US21079	20000802

AU 2000065118 A	AU 2000-65118	20000802
EP 1204424 A1	EP 2000-952415	20000802
	WO 2000-US21079	20000802

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000065118 A		Based on	WO 200108703
EP 1204424	A1	Based on	WO 200108703

PRIORITY APPLN. INFO: US 1999-368076 19990803
AN 2001-191424 [19] WPIDS
AB WO 200108703 A UPAB: 20010405
NOVELTY - Isolated and infectious **respiratory syncytial virus** (**RSV**) particle (A) comprising an **RSV** (anti)genome that has at least one functional deletion in a virus accessory gene (I).

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a vaccine containing an **RSV**, the genome of which contains the reverse complement of a mRNA-encoding sequence linked to a polymerase-binding site (PBS) of an **RSV**.

ACTIVITY - Antiviral.
No data given.

MECHANISM OF ACTION - (A) Induce both humoral and cellular immune responses.

USE - (A) are useful for preparing attenuated, live vaccines, including those that express heterologous gene products (particularly from another strain of **RSV**, some other virus or pathogen, cellular protein or tumor antigen). Also negative-strand **RSV** RNA templates can be used to express heterologous gene products (e.g. viral proteins or ribozymes for prevention or treatment of disease) in cells and/or to rescue heterologous genes in virus particles.

ADVANTAGE - **RSV** has extensive strain variability, so any of many thousand **variants** can be chosen for construction of chimeric viruses, avoiding the problem of host resistance encountered with e.g. vaccinia. Contrary to earlier reports, the M2-1 expression plasmid is not essential for production of infectious **RSV** from cDNA, rather the N, P and L proteins (of the polymerase complex) are sufficient to produce an attenuated particle.
Dwg.0/25

L24 ANSWER 26 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2001-103088 [11] WPIDS
DOC. NO. CPI: C2001-030283
TITLE: Isolated chimeric **human**-bovine **respiratory syncytial virus** (**RSV**), useful in an attenuated vaccine to elicits an immune response against either or both **human RSV** A or **RSV** B.
DERWENT CLASS: B04 D16
INVENTOR(S): BUCHHOLZ, U; COLLINS, P L; KREMPLE, C D; MURPHI, B R; WHITEHEAD, S S; KREMPL, C D; MURPHY, B R
PATENT ASSIGNEE(S): (USSH) US DEPT HEALTH & HUMAN SERVICES
COUNTRY COUNT: 94
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001004335	A2	20010118	(200111)*	EN	148

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ
NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM DZ
EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK
LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI
SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2000056415 A 20010130 (200127)
BR 2000013195 A 20020723 (200257)
EP 1287152 A2 20030305 (200319) EN
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
KR 2002092343 A 20021211 (200328)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001004335	A2	WO 2000-US17755	20000623
AU 2000056415	A	AU 2000-56415	20000623
BR 2000013195	A	BR 2000-13195	20000623
		WO 2000-US17755	20000623
EP 1287152	A2	EP 2000-941756	20000624
		WO 2000-US17755	20000624
KR 2002092343	A	KR 2002-700318	20020109

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000056415	A	Based on	WO 200104335
BR 2000013195	A	Based on	WO 200104335
EP 1287152	A2	Based on	WO 200104335

PRIORITY APPLN. INFO: US 1999-143132P 19990709
AN 2001-103088 [11] WPIDS
AB WO 200104335 A UPAB: 20030214
NOVELTY - An isolated chimeric **human**-bovine **respiratory syncytial virus** (**RSV**) that is infectious and attenuated in **humans**, is new.
DETAILED DESCRIPTION - An isolated chimeric **human**-bovine **respiratory syncytial virus** (**RSV**) that is infectious and attenuated in **humans**, is new.
The virus comprises a major nucleocapsid protein (N), a nucleocapsid phosphoprotein (P), a large polymerase protein (L), a RNA polymerase elongation factor, and a partial or complete **RSV** background genome, or antigenome of a **human RSV** or bovine **RSV**, combined with one or more heterologous gene(s) or genome segment(s) of a different **RSV** to form a **human**-bovine chimeric **RSV** genome or antigenome.
INDEPENDENT CLAIMS are also included for the following:
(1) a method (M1) for stimulating the immune system of an individual to induce protection against **RSV**, comprising administering an immunologically sufficient amount of the chimeric **RSV**;
(2) an isolated **polynucleotide** comprising a chimeric **RSV** genome or antigenome which includes a partial or complete **RSV** background genome or antigenome of a **human** or bovine **RSV** combined with one or more heterologous gene(s) or genome segment(s) of a different **RSV** to form a **human**-bovine chimeric **RSV** genome or antigenome; and
(3) a method (M2) for producing an infectious attenuated chimeric **RSV** particle from one or more isolated **polynucleotide**

molecules encoding the **RSV**, comprising expressing **RSV** N, P, L and RNA polymerase elongation factor proteins, and an expression vector comprising the **polynucleotide** of (2) in a cell or cell-free lysate.

ACTIVITY - Antiviral.

Young chimpanzees which were determined to be seronegative for **human RSV** were inoculated by both the intranasal and intratracheal routes with a dose of 107 pfu (plaque forming units) per ml of rBRSV or rBRSV/A2 at each site. Each virus was administered to two chimpanzees. Following inoculation of the virus, nasopharyngeal swab samples were taken daily on days 1-10 and 12, and tracheal lavage samples were taken on days 2, 5, 6, 8 and 12. Specimens were frozen and **RSV** titers were measured later by plaque assay on HEp-2 cells. The amount of rhinorrhea, a measure of upper respiratory tract illness, was estimated daily and assigned a score of 0-4 (0=none, 1= trace, 2= mild, 3= moderate, 4= severe). The results were compared to historic controls of animals which had received:

(i) 104 pfu of **recombinant human RSV** strain A2 wild type virus per site (Whitehead, et al., J. Virol. 72:4467-4471, 1998) or

(ii) 105 pfu of the live-attenuated rA2cp28/404 strain A2 vaccine candidate per site (Whitehead, et al., J. Virol. 73:343 8-3442, 1999), administered by the same routes.

Wild type **human RSV** was highly permissive in seronegative chimpanzees, and in this exercise replicated to peak mean titers of more than 4.5 log10 pfu per mI of nasal swab or tracheal lavage sample. The peak rhinorrhea score was 2.5. The live- attenuated vaccine candidate rA2cp248/404 (see, e.g., U.S. Patent No. 5,993,824, issued November 30, 1999; International Publication No. WO 98102530; Collins, et al., Proc Natl. Acad. Sci. USA 92:11563-11567,1995; Whitehead, et al., Virology 247:232-239, 1998) replicated to mean peak titers of 2.5 and 1.4 log10 pfu per mI of swab/lavage in the upper and lower respiratory tracts, respectively, and had a peak rhinorrhea score of 0.8. In contrast, there was no detectable replication of **recombinant** bovine (rBRSV) in either the upper or lower respiratory tracts and no evidence of disease. Thus, even when administered at 100-1000 times the dose of **human RSV**, rBRSV was highly restricted for replication in chimpanzees. The rBRSV/A2 chimera exhibited replication over several days in both the upper and lower respiratory tract.

The shedding was not detected until day 3 or 5 indicates that it was not carryover from the inoculation, as does the length of time over which virus was recovered. The titers were much lower than observed for wild type **human RSV** and moderately lower than observed for the rA2cp248/404 vaccine candidate. These results indicate that the chimeric virus was highly attenuated. Thus, replacement of the G and F glycoprotein genes of rBRSV with their **human RSV** counterparts, which transferred the major antigenic determinants, confers improved growth in chimpanzees while other bovine **RSV** genes contribute to a highly attenuated phenotype.

MECHANISM OF ACTION - Immunostimulant; Anti-**RSV** vaccine.

USE - The chimeric **RSV** is useful in an attenuated vaccine to elicits an immune response against either or both **human RSV** A or **RSV** B (claimed).

Dwg.0/13

L24 ANSWER 27 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2001-103086 [11] WPIDS
DOC. NO. CPI: C2001-030281
TITLE: Isolated infectious **recombinant respiratory syncytial virus** (

RSV) has a modified genome and is used as a noninfectious subunit vaccine and for the production of viral proteins in cell culture.

DERWENT CLASS: B04 D16
INVENTOR(S): BERMINGHAM, A; COLLINS, P L; MURPHY, B R
PATENT ASSIGNEE(S): (USSH) US DEPT HEALTH & HUMAN SERVICES
COUNTRY COUNT: 94
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001004321	A1	20010118	(200111)*	EN	124

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

AU 2000059181	A	20010130	(200127)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001004321	A1	WO 2000-US18534	20000707
AU 2000059181	A	AU 2000-59181	20000707

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000059181	A	Based on	WO 200104321

PRIORITY APPLN. INFO: US 1999-143097P 19990709
AN 2001-103086 [11] WPIDS
AB WO 200104321 A UPAB: 20010224

NOVELTY - Isolated infectious **recombinant respiratory syncytial virus** (**RSV**) (I) comprises an **RSV** genome or antigenome, major nucleocapsid (N) protein, nucleocapsid phosphoprotein (P), large polymerase protein (L) and RNA polymerase elongation factor and has a modification in the genome/antigenome of the second translational open reading frame encoded by the M2 gene (M2 ORF2).

DETAILED DESCRIPTION - Isolated infectious **recombinant respiratory syncytial virus** (**RSV**) (I) comprises an **RSV** genome or antigenome, major nucleocapsid (N) protein, nucleocapsid phosphoprotein (P), large polymerase protein (L) and RNA polymerase elongation factor and has a modification in the genome/antigenome which is complete or partial deletion of the second translational open reading frame encoded by the M2 gene (M2 ORF2) or at least one nucleotide change to reduce/ablate M2 ORF2 expression.

INDEPENDENT CLAIMS are also included for the following:

(1) an isolated **polynucleotide** molecule (II) comprising a **RSV** genome or antigenome modified by a partial or complete deletion of M2 ORF2 or one or more nucleotide changes that reduce or ablate expression of M2 ORF;

(2) a method for producing an infectious attenuated **RSV** particle from one or more isolated **polynucleotide** molecules encoding the **RSV**;

(3) an isolated infectious **recombinant RSV** (III)

comprising an **RSV** genome or antigenome, major nucleocapsid (N) protein, nucleocapsid phosphoprotein (P), large polymerase protein (L) and RNA polymerase elongation factor and an amino acid substitution at Asn43 of the **RSV** polymerase gene L; and

(4) a method for producing one or more purified **RSV** proteins comprising infecting a host cell permissive of **RSV** infection with a **recombinant RSV** that has an M2 ORF deletion or knock-out mutation in its genome or antigenome, isolating the **recombinant RSV** from the host cell and purifying the one or more **RSV** proteins.

ACTIVITY - Immunostimulant; respiratory general; antiinflammatory.

MECHANISM OF ACTION - Vaccine; gene therapy.

Recombinant RSV unable to express NS 1 (rA2 Delta NS1) or M2-2 (rA2 Delta M2-2) viruses were administered individually to juvenile **RSV**-seronegative chimpanzees by combined intranasal and intratracheal inoculation at 105 pfu per ml per site. Nasopharyngeal swabs and tracheal lavage samples were collected at intervals over 10 days post infection and assayed for virus titer to monitor virus replication. The mean peak titer for the nasopharyngeal swab was 5 for wild type **RSV**, 1.6 for rA2 Delta NS1 and 1.5 for rA2 Delta M2-2, for the tracheal lavage the mean peak titer was 5.5 for wild type **RSV**, 1.2 for rA2 Delta NS1 and less than 0.7 for rA2 Delta M2-2. The chimpanzees were monitored daily for rhinorrhea, a symptom of upper respiratory tract illness and the mean peak score determined for each group. For the wild type **RSV** the score was 3 (moderate), for rA2 Delta NS1 it was 2 (mild) and for rA2 Delta M2-2 it was 1.8.

USE - (I) elicits a protective immune response to **RSV** in a vaccinated host (claimed). This immune response is protective against serious lower respiratory tract disease e.g. pneumonia and bronchioloitis when the individual is subsequently infected with wild type **RSV**. (I) is administered to an individual seronegative for antibodies to **RSV** or possessing transplacentally acquired maternal antibodies to **RSV**. (I) elicits an immune response against **human RSV** A and/or **RSV** B (claimed). (I) can be used for the production of viral proteins in cell culture.

The M2 ORF2 deletion or knockout **mutant** is also used as a vector for transient gene therapy of the respiratory tract. The vector incorporates a sequence encoding a product of interest e.g. cytokines such as interleukin 2 (IL-2), IL-4, interferon gamma (IF-gamma) and granulocyte-macrophage colony stimulating factor (GM-CSF).

ADVANTAGE - Previously a chemotherapeutic agent ribavirin and pooled donor IgG has been used to treat **human RSV** but these methods lack long-term effectiveness and are inappropriate for widespread use.

Dwg.0/6

L24 ANSWER 28 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2001-081053 [09] WPIDS
DOC. NO. CPI: C2001-023408
TITLE: Isolated **human**-bovine chimeric **parainfluenza** virus (PIV), useful in an attenuated vaccine to elicits an immune response against one or more virus(es) selected from **human** PIV1 (HPIV1), HPIV2 and HPIV3.
DERWENT CLASS: B04 D16
INVENTOR(S): BAILLY, J E; COLLINS, P L; DURBIN, A P; MURPHY, B R; SCHMIDT, A C; SKIADOPOULOS, M H
PATENT ASSIGNEE(S): (USSH) US DEPT HEALTH & HUMAN SERVICES
COUNTRY COUNT: 94
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001004320	A1	20010118	(200109)*	EN	148
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2000056303	A	20010130	(200127)		
EP 1194564	A1	20020410	(200232)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
BR 2000013190	A	20020716	(200255)		
KR 2002022768	A	20020327	(200264)		
CN 1369011	A	20020911	(200282)		
JP 2003504064	W	20030204	(200320)		170

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001004320	A1	WO 2000-US17066	20000616
AU 2000056303	A	AU 2000-56303	20000616
EP 1194564	A1	EP 2000-941614	20000616
		WO 2000-US17066	20000616
BR 2000013190	A	BR 2000-13190	20000615
		WO 2000-US17066	20000615
KR 2002022768	A	KR 2002-700325	20020109
CN 1369011	A	CN 2000-810120	20000616
JP 2003504064	W	WO 2000-US17066	20000616
		JP 2001-509524	20000616

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000056303	A	Based on	WO 200104320
EP 1194564	A1	Based on	WO 200104320
BR 2000013190	A	Based on	WO 200104320
JP 2003504064	W	Based on	WO 200104320

PRIORITY APPLN. INFO: US 1999-143134P 19990709
AN 2001-081053 [09] WPIDS
AB WO 200104320 A UPAB: 20021105
NOVELTY - An isolated **human**-bovine chimeric **parainfluenza** virus (PIV) that is infectious and attenuated in **humans**, is new.

DETAILED DESCRIPTION - An isolated **human**-bovine chimeric **parainfluenza** virus (PIV) that is infectious and attenuated in **humans**, is new.

The virus comprises a major nucleocapsid protein (N), a nucleocapsid phosphoprotein (P), a large polymerase protein (L), and a partial or complete PIV background genome, or antigenome of a **human** PIV (HPIV) or bovine PIV (BPIV), combined with one or more heterologous gene(s) or genome segment(s) of a different PIV to form a **human**-bovine chimeric PIV genome or antigenome.

INDEPENDENT CLAIMS are also included for the following:

(1) a method for stimulating the immune system of an individual to

induce protection against PIV, comprising administering an immunologically sufficient amount of the chimeric PIV;

(2) an isolated **polynucleotide** comprising a chimeric PIV genome or antigenome which includes a partial or complete PIV background genome or antigenome of a **human** or bovine PIV combined with a heterologous gene or genome segment of a different PIV to form a **human**-bovine chimeric PIV genome or antigenome;

(3) a method for producing an infectious attenuated chimeric PIV particle from one or more isolated **polynucleotide** molecules encoding the PIV, comprising expressing PIV N, P, and L proteins, and an expression vector comprising the **polynucleotide** of (2) in a cell or cell-free lysate; and

(4) an expression vector comprising an operably linked transcriptional promoter, the **polynucleotide** sequence of (2) and a transcriptional terminator.

ACTIVITY - Antiviral.

The rJS (wild-type HPIV3), Ka parent (Kansas BPIV3 strain), cKa (chimeric Ka strain), SF parent (Shipping fever BPIV3 strain) and cSF (chimeric SF strain) were administered intranasally and intratracheally at a dose of 100000 TCID50 per site to rhesus monkeys. Replication was monitored using standard procedures for obtaining samples from the upper (nasopharyngeal swab specimens) and lower (tracheal lavage specimens) respiratory tract and for titering the virus in LLC-MK2 cells. The cKa and cSF **recombinants** were significantly attenuated for the upper respiratory tract exhibiting, respectively, a 63-fold or a 32-fold reduction in mean peak virus titer compared to that of the rJS HPIV3 parent. Both cKa and cSF were also attenuated for the lower respiratory tract, but this difference was only statistically significant for cSF. The low level of replication of rJS in the lower respiratory tract made it difficult to demonstrate in a statistically-significant fashion further restriction of replication due to an attenuation phenotype at this site.

The level of replication of each chimeric virus, cKa and cSF, was not significantly different from its bovine parent in the upper or the lower respiratory tract, although the chimeric viruses each replicated better than their BPIV3 parents in the upper respiratory tract.

MECHANISM OF ACTION - Anti-PIV vaccine.

USE - The chimeric PIV is useful in an attenuated vaccine to elicits an immune response against one or more virus(es) selected from HPIV1, HPIV2 and HPIV3.

Preferably, the chimeric PIV elicits an immune response against HPIV3 and another virus selected from HPIV1, HPIV2 or HPIV3 (claimed).

Dwg.0/11

L24 ANSWER 29 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2001-091926 [10] WPIDS
DOC. NO. CPI: C2001-027208
TITLE: **Recombinant respiratory syncytial virus (RSV)** incorporating a heterologous **polynucleotide** encoding an immune modulatory molecule is used as a vaccine to provide an immune response to **RSV**.
DERWENT CLASS: B04 D16
INVENTOR(S): BURKREYEV, A; COLLINS, P L; MURPHY, B R; WHITEHEAD, S S; BUKREYEV, A
PATENT ASSIGNEE(S): (USSH) US DEPT HEALTH & HUMAN SERVICES
COUNTRY COUNT: 95
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG

WO 2001004271 A2 20010118 (200110)* EN 154
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2000062112 A 20010130 (200127)
EP 1194581 A2 20020410 (200232) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI
BR 2000013202 A 20020924 (200272)
CN 1384883 A 20021211 (200324)
KR 2002092889 A 20021212 (200328)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001004271	A2	WO 2000-US19042	20000712
AU 2000062112	A	AU 2000-62112	20000712
EP 1194581	A2	EP 2000-948641	20000712
		WO 2000-US19042	20000712
BR 2000013202	A	BR 2000-13202	20000712
		WO 2000-US19042	20000712
CN 1384883	A	CN 2000-810303	20000712
KR 2002092889	A	KR 2002-700505	20020114

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000062112	A	Based on	WO 200104271
EP 1194581	A2	Based on	WO 200104271
BR 2000013202	A	Based on	WO 200104271

PRIORITY APPLN. INFO: US 1999-143425P 19990713
AN 2001-091926 [10] WPIDS
AB WO 200104271 A UPAB: 20010220
NOVELTY - Infectious **recombinant respiratory syncytial virus** (**RSV**) (I) comprising a **recombinant RSV** genome or antigenome incorporating a heterologous **polynucleotide** encoding an immune modulatory molecule, a major nucleocapsid (N) protein, nucleocapsid phosphoprotein (P), large polymerase protein (L) and a RNA polymerase elongation factor, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated **polynucleotide** molecule (II) comprising a **RSV** genome or antigenome modified to incorporate a **polynucleotide** sequence encoding an immune modulatory molecule; and

(2) a method for producing an infectious attenuated **RSV** particle from one or more isolated **polynucleotide** molecules encoding the **RSV**.

ACTIVITY - Immunostimulator.

Balb/c mice were infected intranasally with 106 plaque forming units (pfu) rRSV/mIFN gamma , rRSV/chloramphenicol acetyl transferase (CAT) or wt **RSV**. Serum samples were collected on days 0, 28 and 56 and analyzed by **RSV**-specific and antibody isotype-specific enzyme

linked immunosorbent assay and by an **RSV** neutralization assay. The levels of IgA antibodies induced by the viruses were not significantly different, there was a significant increase, four fold, in total IgG specific to **RSV** F protein in mice vaccinated with rRSV/mIFN gamma compared to animals vaccinated with wt **RSV** or **RSV** /CAT on day 56 but not on day 28. Neutralizing antibody titers of mice infected with rRSV/mIFN gamma compared with wt **RSV** and **RSV**/CAT were lower on day 28 but modestly higher on day 56.

MECHANISM OF ACTION - Vaccine.

USE - (I) elicits a protective immune response to **RSV** in a vaccinated host (claimed). (I) is administered to an individual seronegative for antibodies to **RSV** or possessing transplacentally acquired maternal antibodies to **RSV**. (I) elicits an immune response against **human RSV** A and/or **RSV** B.

ADVANTAGE - (I) induces titers of serum Immunoglobulin G (IgG) that are at least 2-10 fold higher than levels of serum IgG induced by wt **RSV**.

Previously a chemotherapeutic agent ribavirin and pooled donor IgG has been used to treat **RSV** but these methods lack long-term effectiveness and are inappropriate for widespread use.

Dwg.0/7

L24 ANSWER 30 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2001-071448 [08] WPIDS
CROSS REFERENCE: 2002-520377 [56]
DOC. NO. CPI: C2001-020057
TITLE: Obtaining an attenuated vaccine comprising **recombining** nucleic acids that comprise a complete or partial genomic library of a virus or cell and screening to identify those that are attenuated, useful for treating viral infections.
DERWENT CLASS: B04 D16
INVENTOR(S): APT, D; DELCARDAYRE, S; HOWARD, R; PUNNONEN, J; STEMMER, W P C
PATENT ASSIGNEE(S): (MAXY-N) MAXYGEN INC
COUNTRY COUNT: 95
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001000234	A2	20010104	(200108)*	EN	117
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2000058809	A	20010131	(200124)		
EP 1196552	A2	20020417	(200233)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
JP 2003503039	W	20030128	(200309)		149

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001000234	A2	WO 2000-US16984	20000620
AU 2000058809	A	AU 2000-58809	20000620

EP 1196552	A2	EP 2000-944760	20000620
		WO 2000-US16984	20000620
JP 2003503039	W	WO 2000-US16984	20000620
		JP 2001-505941	20000620

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000058809	A	Based on	WO 200100234
EP 1196552	A2	Based on	WO 200100234
JP 2003503039	W	Based on	WO 200100234

PRIORITY APPLN. INFO: US 1999-344655 19990625
AN 2001-071448 [08] WPIDS
CR 2002-520377 [56]
AB WO 200100234 A UPAB: 20030206
NOVELTY - A method (M1) for obtaining an attenuated vaccine comprising **recombining** first nucleic acids that comprise a complete or partial genomic library of a virus or cell with a second set and screening to identify those that are attenuated, is new.

DETAILED DESCRIPTION - A method (M1) of obtaining an attenuated vaccine comprises:

(a) **recombining** a first set of nucleic acid segments that comprises a complete or partial genomic library of a cell with a second set of nucleic acid segments to form a library of **recombinant** nucleic acid fragments;

(b) screening viruses or cells that contain members of the library of **recombinant** nucleic acid fragments to identify those viruses or cells that are attenuated under physiological conditions that exist in a host organism; and

(c) screening the attenuated viruses or cells to identify those that can induce an immune response against a pathogenic agent that displays an immunogenic determinant that is also displayed by the attenuated viruses or cells.

INDEPENDENT CLAIMS are also included for the following:

(1) an attenuated virus or cell obtained by M1;

(2) a vaccine composition comprising the virus or cell of (1);

(3) a method (M2) for vaccinating an animal comprising administering the composition of (2);

(4) a method (M3) for obtaining a chimeric attenuated vaccine comprising:

(a) **recombining** a first set of one or more nucleic acid segments from a virus or cell with a second set of one or more nucleic acid segments, where the nucleic acid segments of the second set confer upon viruses or cells that contain the nucleic acid segments a property that is desirable for vaccination, to form a library of **recombinant** DNA fragments;

(b) identifying attenuated viruses or cells by screening viruses or cells that contain members of the library of **recombinant** DNA fragments to identify those viruses or cells that are attenuated under physiological conditions present in a host organism inoculated with the viruses or cells; and

(c) screening the attenuated viruses or cells to identify those that exhibit an improvement in the property that is desirable for vaccination;

(5) a chimeric attenuated vaccine that comprises an attenuated virus or cell obtained by M3;

(6) a vaccine composition comprising an attenuated virus or cell of (5); and

(7) a method (M4) of vaccinating an animal comprising administering

the composition of (6).
ACTIVITY - Antiviral; antibacterial; antiparasitic.
No biological data is given.
MECHANISM OF ACTION - Vaccine.
No biological data is given.
USE - The methods are useful for producing engineered attenuated vaccines which can be used against pathogenic agents such as viruses, bacteria, and parasites.
ADVANTAGE - The vaccines have improved expression of an immunogenic polypeptide, improved specific uptake, enhanced stability and enhanced immunogenecity.
Dwg.0/6

L24 ANSWER 31 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2001-638503 [73] WPIDS
CROSS REFERENCE: 2002-121021 [16]
DOC. NO. CPI: C2001-188830
TITLE: New expression vector, useful for improving expression of transgene or polypeptide, comprises 3 **polynucleotides** encoding crippled selectable marker, heterologous polypeptide or second amplifiable selectable marker.
DERWENT CLASS: B04 D16
INVENTOR(S): INNIS, M; SCOTT, E M
PATENT ASSIGNEE(S): (CHIR) CHIRON CORP
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2001024807	A1	20010927	(200173)*		27
US 6451539	B2	20020917	(200264)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
US 2001024807	A1	Provisional	US 1999-162930P	19991101
		Div ex	US 1999-475460	19991230
			US 2000-748061	20001222
US 6451539	B2	Provisional	US 1999-162930P	19991101
		Div ex	US 1999-475460	19991230
			US 2000-748061	20001222

FILING DETAILS:

PATENT NO	KIND		PATENT NO
US 6451539	B2	Div ex	US 6316253

PRIORITY APPLN. INFO: US 1999-162930P 19991101; US 1999-475460 19991230; US 2000-748061 20001222
AN 2001-638503 [73] WPIDS
CR 2002-121021 [16]
AB US2001024807 A UPAB: 20021031
NOVELTY - A new expression vector comprising:
(a) a first **polynucleotide** encoding a first, crippled selectable marker;
(b) a second **polynucleotide** encoding a heterologous polypeptide of interest; and

(c) a third **polynucleotide** encoding a second amplifiable selectable marker, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a plasmid selected from pESN1dhfr, pESN2dhfr, pESN2dhfr, pneo1dhfr5'del, pneo2dhfr5'del, pneo3dhfr5'del, and pdhfr3'del;

(2) producing a polypeptide of interest in a host cell;

(3) a host cell line that produces a polypeptide of interest;

(4) a transfection system comprising:

(a) a first construct comprising in a suitable backbone, a sequence encoding a first selectable marker and a sequence encoding a second selectable marker containing at least one disabling mutation in its coding sequence; and

(b) a second construct comprising in a suitable backbone, a **polynucleotide** sequence of interest and a sequence encoding a third selectable marker which is the same as the second selectable marker except that the third marker contains at least one disabling mutation in a different region of the coding sequence than the second marker;

(5) producing a mammalian cell line for expression of a selected **polynucleotide** sequence;

(6) a mammalian cell produced from (5);

(7) producing a polypeptide of interest in a host mammalian cell; and

(8) a mammalian cell line that produces a polypeptide of interest, where the cell line is produced from (7).

USE - The expression vectors are useful for the efficient expression of desired polypeptides or improving expression of a transgene of interest. The transformed cells can be used in the preparation of continuous cell lines in which the cells are essentially immortal or for the preparation of established cell lines that have the potential to be subcultured in vitro.

Dwg.0/10

L24 ANSWER 32 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2001-496169 [54] WPIDS
CROSS REFERENCE: 2001-090481 [08]
DOC. NO. CPI: C2001-148944
TITLE: New DNA fragmentation factor polypeptides and **polynucleotides**, useful for inhibiting the growth of cancer cells, as well as for inducing apoptosis of cells.
DERWENT CLASS: B04 D16
INVENTOR(S): LIU, X; WANG, X
PATENT ASSIGNEE(S): (TEXA) UNIV TEXAS SYSTEM
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2001011078	A1	20010802	(200154)*		56

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2001011078	A1 Div ex	US 1998-61702	19980416
		US 2000-748451	20001222

FILING DETAILS:

PATENT NO	KIND	PATENT NO

US 2001011078 A1 Div ex US 6165737

PRIORITY APPLN. INFO: US 1998-61702 19980416; US 2000-748451 20001222

AN 2001-496169 [54] WPIDS

CR 2001-090481 [08]

AB US2001011078 A UPAB: 20010924

NOVELTY - An isolated polypeptide, designated DNA fragmentation factor 40 (DFF40), is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated peptide having 10-50 consecutive residues of a DFF40 DNA fragmentation factor;

(2) an isolated oligonucleotide of 15-50 consecutive bases of a nucleic acid, or its complement, encoding a DFF40 DNA fragmentation factor;

(3) a hybridoma cell that produces a monoclonal antibody that binds immunologically to a DFF40 DNA fragmentation factor;

(4) a polyclonal antisera, antibodies of which bind immunologically to a DFF40 DNA fragmentation factor;

(5) an isolated nucleic acid comprising a region, or its complement, encoding a DFF40 DNA fragmentation factor or its allelic **variant** ;

(6) a plasmid construct comprising a first nucleic acid encoding a DFF40 DNA fragmentation factor;

(7) inducing apoptosis in a cell comprising providing the cell with a DFF40 DNA fragmentation factor, where the provision of the DFF40 to the cell results in apoptosis;

(8) inhibiting the growth of a cancer cell by contacting a cancer cell with a DFF40 DNA fragmentation factor under conditions permitting the uptake of the DNA fragmentation factor by the cell, where the presence of the DFF40 in the cell induces apoptosis;

(9) treating cancer comprising contacting a tumor cell within a subject with a nucleic:

(a) encoding a DFF40 DNA fragmentation factor; and

(b) a promoter active in the tumor cell, where the promoter is operably linked to the region encoding the DNA fragmentation factor, under conditions permitting the uptake of the nucleic acid by the tumor cell;

(10) identifying a modulator of DFF40 activity comprising:

(a) providing a cell expressing a DFF40/DFF45 complex;

(b) contacting the cell with a candidate substance;

(c) activating DFF40; and

(d) comparing the apoptosis of the cell in step (c) with the apoptosis observed when the candidate substance is not added, where an alteration in apoptosis indicates that the candidate substance is a modulator the apoptotic activity;

(11) a modulator of apoptotic activity identified by:

(a) providing a cell expressing a DFF40/DFF45 complex;

(b) contacting the cell with a candidate substance;

(c) activating DFF40; and

(d) comparing the apoptosis of the cell in step (c) with the apoptosis observed when the candidate substance is not added, where an alteration in apoptosis indicates that the candidate substance is a modulator of apoptotic activity;

(12) an isolated DNA fragmentation factor complex for regulating chromatin stability, the complex comprising a DFF40 polypeptide and a DFF45 polypeptide;

(13) producing a functional DNA fragmentation factor, comprising:

(a) providing to cell nucleic acids encoding DFF40 and DFF45

polypeptides; and
(b) expressing the complex in a cell, where the coexpression of the polypeptide allows for the formation of a functional DFF40 polypeptide.
ACTIVITY - Cytostatic; immunostimulant.
No biological data is given.
MECHANISM OF ACTION - Apoptosis modulator; protein therapy.
USE - The polypeptide and polynucleotide are useful for inhibiting the growth of cancer cells, and for inducing apoptosis of cells.
Dwg.0/1

L24 ANSWER 33 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2001-217929 [22] WPIDS
CROSS REFERENCE: 1997-385326 [35]; 1997-393271 [36]; 2000-586352 [52]
DOC. NO. CPI: C2001-064957
TITLE: Producing **human** insulin involves transforming secretory host cell with exogenous **polynucleotide** comprising gene encoding insulin, and culturing host cell.
DERWENT CLASS: B04 D16
INVENTOR(S): CLARK, S A; HALBAN, P; KRUSE, F; NEWGARD, C B; NORMINGTON, K D; QUAADE, C; THIGPEN, A E
PATENT ASSIGNEE(S): (BETA-N) BETAGENE INC; (TEXA) UNIV TEXAS SYSTEM
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 6194176	B1	20010227	(200122)*		107

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
US 6194176	B1	CIP of	US 1996-589028	19960119
			US 1997-785271	19970117

PRIORITY APPLN. INFO: US 1997-785271 19970117; US 1996-589028 19960119
AN 2001-217929 [22] WPIDS
CR 1997-385326 [35]; 1997-393271 [36]; 2000-586352 [52]
AB US 6194176 B UPAB: 20010421
NOVELTY - Producing **human** insulin (HI), comprising transforming secretory host cell (C) with an exogenous **polynucleotide** (I) comprising a gene encoding HI under the control of a promoter active in eukaryotic cells, and culturing (C) so that (I) expresses HI, is new. (C) secretes 200-1000 ng of HI/106 cells/hour.
DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a secretory host cell (C) comprising (I) comprising, where (C) secretes 200-1000 ng of HI/106 cells/hour.
ACTIVITY - Antidiabetic.
MECHANISM OF ACTION - Vaccine.
No biological data is given.
USE - The method is useful for the large scale production of heterologous polypeptides and for providing a polypeptide to an animal. (C) is useful in the treatment of diabetes, and in vitro to produce large amount of protein, in vivo to supply therapeutic protein, or in vivo to immunize hosts, for e.g. in the production of monoclonal antibodies.
Dwg.0/24

L24 ANSWER 34 OF 67 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2001379346 MEDLINE
DOCUMENT NUMBER: 21329463 PubMed ID: 11435561
TITLE: Functional analysis of **recombinant respiratory syncytial virus deletion mutants** lacking the **small hydrophobic** and/or attachment glycoprotein gene.
AUTHOR: Techaarpornkul S; Barretto N; Peeples M E
CORPORATE SOURCE: Department of Immunology/Microbiology, Rush-Presbyterian-St. Luke's Medical Center, Chicago, Illinois 60612, USA.
CONTRACT NUMBER: AI47213 (NIAID)
SOURCE: JOURNAL OF VIROLOGY, (2001 Aug) 75 (15) 6825-34.
Journal code: 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200108
ENTRY DATE: Entered STN: 20010827
Last Updated on STN: 20010827
Entered Medline: 20010823

AB **Respiratory syncytial virus** (**RSV**) produces three envelope glycoproteins, the attachment glycoprotein (G), the fusion (F) protein, and the small hydrophobic (SH) protein. It had been assumed, by analogy with other paramyxoviruses, that the G and F proteins would be required for the first two steps of viral entry, attachment and fusion. However, following repeated passage in cell culture, a viable **mutant RSV** that lacked both the G and SH genes was isolated (R. A. Karron, D. A. Buonagurio, A. F. Georgiu, S. S. Whitehead, J. E. Adamus, M. L. Clements-Mann, D. O. Harris, V. B. Randolph, S. A. Udem, B. R. Murphy, and M. S. Sidhu, Proc. Natl. Acad. Sci. USA 94:13,961--13,966, 1997). To explore the roles of the G, F, and SH proteins in virion assembly, function, and cytopathology, we have modified the full-length **RSV** cDNA and used it to rescue infectious **RSV** lacking the G and/or SH genes. The three resulting viruses and the parental virus all contain the green fluorescent protein (GFP) gene that serves to identify infected cells. We have used purified, radiolabeled virions to examine virus production and function, in conjunction with GFP to quantify infected cells. We found that the G protein enhances virion binding to target cells but plays no role in penetration after attachment. The G protein also enhances cell-to-cell fusion, presumably via cell-to-cell binding, and enhances virion assembly or release. The presence or absence of the G protein in virions has no obvious effect on the content of F protein or host cell proteins in the virion. In growth curve experiments, the viruses lacking the G protein produced viral titers that were at least 10-fold lower than titers of viruses containing the G protein. This reduction is due in large part to the less efficient release of virions and the lower infectivity of the released virions. In the absence of the G protein, virus expressing both the F and SH proteins displayed somewhat smaller plaques, lower fusion activity, and slower viral entry than the virus expressing the F protein alone, suggesting that the SH protein has a negative effect on virus fusion in cell culture.

L24 ANSWER 35 OF 67 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 2001205152 MEDLINE
DOCUMENT NUMBER: 21103003 PubMed ID: 11172105
TITLE: **Recombinant** bovine **respiratory syncytial virus** with **deletions**

of the G or **SH** genes: G and F proteins bind heparin.
AUTHOR: Karger A; Schmidt U; Buchholz U J
CORPORATE SOURCE: Institute of Molecular Biology, Friedrich-Loeffler-Institutes, Federal Research Centre for Virus Diseases of Animals, Boddenblick 5a, D-17498 Insel Riems, Germany.
SOURCE: JOURNAL OF GENERAL VIROLOGY, (2001 Mar) 82 (Pt 3) 631-40. Journal code: 0077340. ISSN: 0022-1317.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200104
ENTRY DATE: Entered STN: 20010417
Last Updated on STN: 20010417
Entered Medline: 20010412

AB Bovine **respiratory syncytial virus** (BRSV) encodes three transmembrane envelope glycoproteins, namely the small hydrophobic (SH) protein, the attachment glycoprotein (G) and the fusion glycoprotein (F). The BRSV reverse genetics system has been used to generate viable **recombinant** BRSV lacking either the G gene or the SH gene or both genes. The deletion **mutants** were fully competent for multicycle growth in cell culture, proving that, of the BRSV glycoprotein genes, the SH and G genes are non-essential. Virus morphogenesis was not impaired by either of the deletions. The deletion **mutants** were used to study the role of the F glycoprotein and the contributions of SH and G with respect to virus attachment. Attachment mediated by the F protein alone could be blocked by soluble heparin, but not by chondroitin sulphate. Heparin affinity chromatography revealed that both the BRSV G and F glycoproteins have heparin-binding activity, with the affinity of the F glycoprotein being significantly lower than that of G. Therefore, the roles of the BRSV glycoproteins in virus attachment and receptor binding have to be reconsidered.

L24 ANSWER 36 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2001-032162 [04] WPIDS
DOC. NO. CPI: C2001-009918
TITLE: Use of **recombinant** gene delivery vectors, e.g. lentiviral and feline immunodeficiency virus vectors, for treating or preventing retinal diseases of the eye and diseases of the brain associated with lysosomal storage disorders.
DERWENT CLASS: B04 D16
INVENTOR(S): DAVIDSON, B L; DERKSEN, T A; DUBENSKY, T W; GHODSI, A; HETH, J A; JOLLY, D J; SAUTER, S L; STEIN, C S
PATENT ASSIGNEE(S): (CHIR) CHIRON CORP; (HETH-I) HETH J A; (IOWA) UNIV IOWA RES FOUND
COUNTRY COUNT: 94
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000073482	A1	20001207	(200104)*	EN	52
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2000052955	A	20001218	(200118)		

EP 1183384 A1 20020306 (200224) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000073482	A1	WO 2000-US14582	20000526
AU 2000052955	A	AU 2000-52955	20000526
EP 1183384	A1	EP 2000-937832	20000526
		WO 2000-US14582	20000526

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000052955	A	Based on	WO 200073482
EP 1183384	A1	Based on	WO 200073482

PRIORITY APPLN. INFO: US 1999-136527P 19990528
AN 2001-032162 [04] WPIDS
AB WO 200073482 A UPAB: 20011129
NOVELTY - A lentiviral vector (Lv) comprising a 5' lentiviral LTR (long terminal repeat), a tRNA binding site, a packaging signal, a promoter operably linked to a **polynucleotide** encoding a protein deficient in a lysosomal storage disorder of the brain or eye, an origin of second strand DNA synthesis and a 3' lentiviral LTR, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an feline immunodeficiency virus (FIV) vector comprising a 5' FIV LTR, a tRNA binding site, a packaging signal, a **polynucleotide** encoding beta -glucuronidase operably linked to an FIV LTR promoter or promoter element, an origin of second strand DNA synthesis and a 3' FIV LTR;

(2) a lentiviral vector particle comprising the lentiviral vector (Lv);

(3) an FIV vector particle comprising the FIV vector of claim (1);

(4) a host cell transduced with either:

(a) the lentiviral vector particle of claim (2); or

(b) the FIV vector particle of claim (3); and

(5) a method of treating a lysosomal storage disorder of the eye or brain comprising providing the lentiviral or FIV vector particles and transducing a host cell with the particles under conditions where the protein encoded by the **polynucleotide** is expressed.

ACTIVITY - Ophthalmological; neuroprotective.

MECHANISM OF ACTION - Gene therapy; nucleic acid delivery vector.

FIV vectors expressing beta -glucuronidase were generated which were devoid of vif and ORF 2 (FIV beta gluc Delta vif Delta orf2). Vectors were injected into the striatum of beta -glucuronidase deficient mice and animals sacrificed 3 to 18 weeks later and tissues analyzed for transgene expression, enzyme activity and correction of pathology. FIV beta gluc vector-mediated gene transfer resulted in robust levels of expression in the injected hemisphere, 3 weeks after injection of vector. The level of activity did not decline by 18 weeks and there was no evidence of inflammatory infiltrate. In situ RNA analysis for **human** beta -glucuronidase mRNA confirmed that transduction was limited to cells near the injection site and suggests that mRNA and/or virus was not transported to distant cells. Therefore, the extensive distribution of enzyme resulted from secretion by transduced cells followed by distribution via the CSF

(cerebrospinal fluid) and extracellular fluids with uptake by distant cells. Tissues harvested from beta -glucuronidase deficient mice injected into the right striatum with FIV beta gluc vector were also processed for evaluation of lysosomal distension. In tissues from mice sacrificed 3 weeks after gene transfer correction was noted in the ipsilateral striatum, ipsilateral cortex with slight reductions in storage product noted in the contralateral cortex. By 6 weeks there was dramatic restoration of cellular morphology in both hemispheres of the brain.

USE - The lentiviral vector particle of claim (2) and the FIV particle of claim (3) are used in the manufacture of a composition for treating a lysosomal storage disorder of the eye or brain. They are also used in the manufacture of a composition for treating or preventing cell damage in retinal epithelial cells where the cell damage is associated with Sly syndrome. The lentiviral and FIV vector particles may be administered to a subject in need of treatment by transducing the host cell in vivo. The host cell may also be transduced ex vivo and the transduced cell introduced into a vertebrate subject in need of treatment. Other diseases which may be treated include Salla disease, infantile sialic acid storage disease, cystinosis, Morbus Gaucher disease, type 1 sialidosis, Batten's disease, Mucolipidosis Type IV, Niemann-Pick disease and sialidosis.

ADVANTAGE - Previously employed retroviruses such as monkey leukemia virus (MLV) for gene therapy have a limited application and the frequency of transduction of non-replicating cells is low. The present invention provides treatment for a wide variety of retinal and brain diseases.
Dwg.0/5

L24 ANSWER 37 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2001-061513 [07] WPIDS
DOC. NO. CPI: C2001-017061
TITLE: Use of C-CAM1/C-CAM1 cytoplasmic domain or expression construct comprising nucleic acid sequence encoding C-CAM1/C-CAM1 cytoplasmic domain useful for treating cancer, tumor and inhibiting angiogenesis.
DERWENT CLASS: B04 D16
INVENTOR(S): LIN, S; LOGOTHETIS, C; LUO, W
PATENT ASSIGNEE(S): (TEXA) UNIV TEXAS SYSTEM
COUNTRY COUNT: 23
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000073340	A1	20001207	(200107)*	EN	133
RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE					
W: AU CA JP					
AU 2000051667	A	20001218	(200118)		
EP 1183273	A1	20020306	(200224)	EN	
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					
JP 2003501362	W	20030114	(200306)		143
US 6517828	B1	20030211	(200314)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000073340	A1	WO 2000-US14597	20000526
AU 2000051667	A	AU 2000-51667	20000526
EP 1183273	A1	EP 2000-936343	20000526
		WO 2000-US14597	20000526
JP 2003501362	W	WO 2000-US14597	20000526

		JP 2001-500664	20000526
US 6517828	B1 Provisional	US 1999-136563P	19990528
		US 2000-580043	20000526

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2000051667 A	Based on	WO 200073340
EP 1183273	A1 Based on	WO 200073340
JP 2003501362 W	Based on	WO 200073340

PRIORITY APPLN. INFO: US 1999-136563P 19990528; US 2000-580043 20000526

AN 2001-061513 [07] WPIDS

AB WO 200073340 A UPAB: 20011129

NOVELTY - Use (M1) of a C-CAM1 (cell adhesion molecule)/C-CAM1 cytoplasmic domain (I) or an expression construct comprising a nucleic acid sequence encoding (I)/C-CAM1, under the control of promoter operable in eukaryotic cell, for inhibiting hyperproliferative cell growth, inhibiting angiogenesis and for treating tumor by inhibiting angiogenesis around the tumor, or for treating cancer.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a composition comprising (I) isolated away from other C-CAM1 domains.

ACTIVITY - Cytostatic; antitumor.

The effects of C-CAM1 and its **mutant** proteins on the tumorigenicity of DU145 cells in vivo were examined in a nude mouse xenograft model. DU145 cells, which were derived from a **human** prostate cancer that had metastasized to brain, were tumorigenic when injected into nude mice. DU145 cells did not express C-CAM1 message, as assessed by the RNase protection assay with a probe for the cytoplasmic domain of C-CAM1 cDNA. When compared with control parental cells, DU145 cells expressing wild-type C-CAM1 exhibited reduced tumorigenicity. In comparison, cells infected with control viruses that contained the C-CAM1 sequence in the antisense orientation (Ad AS C-CAM1) did not show reduction in tumor incidence or size, suggesting that the reduced tumorigenicity of C-CAM1 infected cells was caused by C-CAM1 expression. The **mutant** lacking the D1 domain (CAM1-AD1) also suppressed the tumorigenicity of DU145 cells, although to a lesser extent. In contrast, **mutants** CAM1-H458 and CAM1-G454, which had shortened cytoplasmic domains, were unable to suppress tumor growth in vivo. These results suggested that C-CAM1-mediated tumor suppressive activity did not require the adhesion domain but does require the cytoplasmic domain.

MECHANISM OF ACTION - Angiogenesis inhibitor; hyperproliferative cell growth inhibitor; gene therapy; interacts with other kinases to elicit negative signal by signal transduction, for cell growth.

USE - C-CAM1/C-CAM1 cytoplasmic domain or nucleic acids encoding C-CAM1/C-CAM1 cytoplasmic domain is useful for inhibiting hyperproliferative cell growth such as a cancer cell, affected by cancers of lung, breast, melanoma, colon, renal, testicular, ovarian, lung, prostate, hepatic, germ cancer, epithelial, prostate, head and neck, pancreatic cancer, glioblastoma, astrocytoma, oligodendroglioma, ependymomas, neurofibrosarcoma, meningia, liver, spleen, lymph node, small intestine, blood cells, colon, stomach, thyroid, endometrium, prostate, skin, esophagus, bone marrow, and blood, inhibiting angiogenesis and for treating tumor by inhibiting angiogenesis around the tumor. A composition comprising a nucleic acid sequence encoding C-CAM1 polypeptide, under the control of a eukaryotic promoter has a bystander effect, in which the composition is administered to a non-cancerous cell in the patient to treat cancer.

ADVANTAGE - Inhibition of angiogenesis with C-CAMs provides a novel and general approach for treating both primary and secondary tumors than current anti-tumor therapies which target only the hyperproliferative tumor cells.
Dwg.0/6

L24 ANSWER 38 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2000-687044 [67] WPIDS
DOC. NO. CPI: C2000-208979
TITLE: Producing attenuated negative stranded RNA virus vaccines from cloned sequences, useful for immunizing against e.g. **respiratory syncytial virus**, **human parainfluenza** virus, Sendai virus Newcastle disease virus, mumps virus and measles virus.
DERWENT CLASS: B04 C06 D16
INVENTOR(S): COLLINS, P L; DURBIN, A P; MURPHY, B R; SKIADOPOULOS, M H
PATENT ASSIGNEE(S): (USSH) US DEPT HEALTH & HUMAN SERVICES
COUNTRY COUNT: 93
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000061737	A2	20001019	(200067)*	EN	136
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2000042315	A	20001114	(200108)		
EP 1171623	A2	20020116	(200207)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
CN 1347458	A	20020501	(200252)		
KR 2002008831	A	20020131	(200254)		
BR 2000011159	A	20020723	(200257)		
JP 2002541798	W	20021210	(200301)		155

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000061737	A2	WO 2000-US9695	20000412
AU 2000042315	A	AU 2000-42315	20000412
EP 1171623	A2	EP 2000-922075	20000412
		WO 2000-US9695	20000412
CN 1347458	A	CN 2000-806224	20000412
KR 2002008831	A	KR 2001-713102	20011013
BR 2000011159	A	BR 2000-11159	20000412
		WO 2000-US9695	20000412
JP 2002541798	W	JP 2000-611661	20000412
		WO 2000-US9695	20000412

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000042315	A	Based on	WO 200061737
EP 1171623	A2	Based on	WO 200061737
BR 2000011159	A	Based on	WO 200061737

JP 2002541798 W Based on WO 200061737

PRIORITY APPLN. INFO: US 1999-129006P 19990413
AN 2000-687044 [67] WPIDS
AB WO 200061737 A UPAB: 20001223
NOVELTY - A method for producing attenuated negative stranded RNA virus vaccines from cloned sequences, is new.

DETAILED DESCRIPTION - A method (I) for producing an isolated, attenuated, **recombinant** negative stranded RNA virus (nsRV) from 1 or more isolated **polynucleotide** molecules encoding the nsRV, comprising co-expressing (in a cell or cell-free system) 1 or more expression vectors which comprise 1 or more **polynucleotide** molecules encoding a **recombinant** genome or antigenome and essential viral proteins necessary to produce an infective virus particle of the nsRV. The **recombinant** genome or antigenome is modified to encode a mutation within a **recombinant** protein of the **recombinant** virus at an amino acid position corresponding to an amino acid position of an attenuating mutation identified in a heterologous, **mutant** nsRV. The mutation, by incorporation within the **recombinant** protein confers an attenuated phenotype on the **recombinant** virus.

INDEPENDENT CLAIMS are also included for the following:

(1) an isolated attenuated **recombinant** nsRV comprising a **recombinant** genome or antigenome and essential viral proteins necessary to produce an infectious particle of the **recombinant** nsRV (the **recombinant** genome or antigenome is modified to encode a mutation within a **recombinant** protein of the virus at an amino acid position corresponding to an amino acid position of an attenuating mutation identified in a heterologous, **mutant** nsRV (the mutation, by incorporation within the **recombinant** protein confers an attenuated phenotype on the **recombinant** virus); and

(2) an expression vector comprising an operably linked transcriptional promoter, a **polynucleotide** molecule encoding a **recombinant** genome or antigenome of a **recombinant** nsRV and a transcriptional terminator (the **recombinant** genome or antigenome is modified to encode a mutation within a **recombinant** protein of the virus at an amino acid position corresponding to an amino acid position of an attenuating mutation identified in a heterologous **mutant** nsRV; the mutation by incorporation within the **recombinant** protein confers an attenuated phenotype on the **recombinant** virus).

ACTIVITY - Antiviral.
No biological data given.
MECHANISM OF ACTION - Vaccine.
USE - The **recombinant** viruses produced may be used for stimulating a patients immune system to induce protection against a negative stranded RNA virus (nsRV) (claimed) such as **respiratory syncytial virus** (**RSV**), especially **human RSV** subgroups A and B, bovine **RSV**, murine **RSV** or avian pneumovirus, **human parainfluenza** virus (HPIV) 1, HPIV2, HPIV 3, bovine PIV (BPIV), Sendai virus (SeV), Newcastle disease virus (NDV), simian virus 5 (SV5), mumps virus (MuV), measles virus (MeV), canine distemper virus (CDV), rabies virus (RaV) or vesicular stomatitis virus (VSV).
Dwg.0/5

L24 ANSWER 39 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2000-679462 [66] WPIDS
DOC. NO. CPI: C2000-206611
TITLE: Infectious chimeric **respiratory**

syncytial virus (**RSV**)
produced from cloned nucleotide sequences, useful as a vaccine against diseases caused by the virus, such as pneumoniae and bronchiolitis.

DERWENT CLASS: B04 D16
INVENTOR(S): COLLINS, P L; MURPHY, B R; WHITEHEAD, S S
PATENT ASSIGNEE(S): (USSH) US DEPT HEALTH & HUMAN SERVICES
COUNTRY COUNT: 92
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000061611	A2	20001019	(200066)*	EN	278
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2000040655	A	20001114	(200108)		
EP 1169457	A2	20020109	(200205)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
KR 2002013526	A	20020220	(200257)		
BR 2000011160	A	20021008	(200277)		
CN 1364195	A	20020814	(200280)		
JP 2002541785	W	20021210	(200301)		309

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000061611	A2	WO 2000-US8802	20000331
AU 2000040655	A	AU 2000-40655	20000331
EP 1169457	A2	EP 2000-920058	20000331
		WO 2000-US8802	20000331
KR 2002013526	A	KR 2001-713099	20011013
BR 2000011160	A	BR 2000-11160	20000331
		WO 2000-US8802	20000331
CN 1364195	A	CN 2000-806217	20000331
JP 2002541785	W	JP 2000-611553	20000331
		WO 2000-US8802	20000331

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000040655	A	Based on	WO 200061611
EP 1169457	A2	Based on	WO 200061611
BR 2000011160	A	Based on	WO 200061611
JP 2002541785	W	Based on	WO 200061611

PRIORITY APPLN. INFO: US 1999-291894 19990413
AN 2000-679462 [66] WPIDS
AB WO 200061611 A UPAB: 20001219
NOVELTY - An isolated infectious chimeric **respiratory syncytial virus** (**RSV**) comprising a major nucleocapsid (N) protein, a nucleocapsid phosphoprotein (P), a large polymerase protein (L), an RNA polymerase elongation factor, and a partial or complete **RSV** genome or antigenome of one **RSV** strain

or subgroup virus combined with a heterologous gene of a different **RSV** strain or subgroup virus, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a method for stimulating the immune system of an individual to induce protection against **RSV** comprising administering the chimeric **RSV**;

(2) an isolated **polynucleotide** molecule comprising a chimeric **RSV** genome or antigenome which includes a partial or complete **RSV** genome or antigenome of one **RSV** strain or subgroup virus combined with a heterologous gene or gene segment of a different **RSV** strain or subgroup virus; and

(3) a method for producing an infectious attenuated chimeric particle from one or more isolated **polynucleotide** molecules encoding the **RSV**, comprising expressing in a cell or cell-free lysate, an expression vector comprising an isolated **polynucleotide** comprising a chimeric **RSV** genome or antigenome and **RSV** N, P, L and RNA polymerase elongation factor proteins.

ACTIVITY - Antiviral.

No relevant biological data is given.

MECHANISM OF ACTION - Vaccine.

No relevant biological data is given.

USE - The chimeric **respiratory syncytial virus** (**RSV**) is useful as a vaccine against **RSV** which causes diseases such as pneumoniae and bronchiolitis in infants.

Dwg.0/27

L24 ANSWER 40 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2000-475771 [41] WPIDS
DOC. NO. CPI: C2000-142631
TITLE: Treating viral infections such as hepatitis B and hepatitis C in mammals, comprises administering a construct encoding interferon omega.
DERWENT CLASS: B04
INVENTOR(S): HORTON, H; PARKER, S
PATENT ASSIGNEE(S): (VICA-N) VICAL INC
COUNTRY COUNT: 21
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000040273	A2	20000713	(200041)*	EN	52

RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE
W: CA JP US

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000040273	A2	WO 1999-US30843	19991228

PRIORITY APPLN. INFO: US 1999-115403P 19990108
AN 2000-475771 [41] WPIDS
AB WO 200040273 A UPAB: 20000831
NOVELTY - A new method of treating a viral infection in a mammal comprises administering a non-infectious integrating construct which comprises a sequence (N1) that encodes interferon omega (IFN omega), or its active **variant**.

DETAILED DESCRIPTION - A new method of treating a viral infection in

a mammal comprises administering a non-infectious integrating construct which comprises a sequence (N1) that encodes interferon omega (IFN omega), or its active **variant**.

N1 is selected from:

(a) a **polynucleotide** that hybridizes under stringent conditions to the 585 nucleotide sequence (I) defined in the specification;

(b) a **polynucleotide** that encodes a polypeptide having an amino acid sequence which, except for at least one but not more than 20 amino acid substitutions, deletions, or insertions, or their combinations, is identical to amino acids -23 to 172, or 1 to 172 of the 195 amino acid sequence defined in the specification (II); or

(c) a **polynucleotide** that encodes a polypeptide comprising residues 86 to 172 of (II).

The **polynucleotide** is expressed in vivo.

ACTIVITY - Antiviral; virucide; hepatotropic; antiinflammatory; antipyretic; cardiant; antibacterial; anti-HIV; cytostatic.

Sprague Dawley rats were injected intramuscularly (i.m.) into the rectus femoris with the plasmid VR4151. This plasmid encodes **human** IFN omega (hIFN omega). Rats received a single i.m. injection of either 0.1 mg (in 100 micro l, 50 micro l bilateral) or 1 mg (in 500 micro l, 250 micro l bilateral) of VR4151. An additional group of rats received 3 consecutive i.m. injections of 1 mg of VR4151 (in 500 micro l, 250 micro l bilateral/day). VR4151 was diluted in 150 mM sodium phosphate buffer, pH 7.2 for the i.m. injections. Serum samples were collected every 2-3 days and assayed in a hIFN omega enzyme linked immunoabsorbant assay (ELISA) (Alexis, San Diego).

Prebleeds of the rats (prior to i.m. injection) were used to determine background hIFN omega levels and were subtracted from the final hIFN omega values obtained at each timepoint after i.m. injection. Each group consisted of 4 rats. Rats injected i.m. once with either 0.1 or 1 mg of VR4151 had an average of 9 and 11 pg/ml hIFN omega , respectively, in the serum by day 6 after injection. By day 9, serum hIFN omega levels were an average of 4 and 12 pg/ml for the groups injected once with either 0.1 or 1 mg VR4151, respectively. Rats injected i.m. with 1 mg VR4151 for 3 consecutive days had an average of 68 pg/ml hIFN omega in the serum by day 5 after the first i.m. injection and 36 pg/ml hIFN omega 4 days later.

MECHANISM OF ACTION - None given.

USE - The construct is used to treat viral infections in mammals, preferably **human** (claimed). Viral diseases which can be treated are hepatitis B (claimed), hepatitis C (claimed), chickenpox, shingles, rubella, influenza, rubeola, mumps, yellow fever, acquired immunodeficiency syndrome (AIDS), mononucleosis, rabies, acute viral gastroenteritis, poliomyelitis, subacute sclerosing panencephalitis, encephalitis, Colorado tick fever, pharyngitis, croup, bronchiolitis, viral pneumonia, pleurodynia, aseptic meningitis, keratitis, conjunctivitis, viral leukemias, polio, myocarditis, hepatitis A, hepatitis D, hepatitis E, and any infections caused by adenoviruses, coxsackieviruses, **parainfluenza** viruses, **respiratory syncytial virus**, reovirus, cytomegalovirus, Epstein-Barr virus, herpes simplex viruses, herpes-zoster- varicella virus, rhinoviruses, rotaviruses, papolomaviruses, enteroviruses, paramyxoviruses, parvoviruses, apthoviruses, Ebola virus, Marburg virus, vesicular stomatitis virus, coronaviruses, Lassa virus, lymphocytic choriomeningitis virus, Machupo virus, Junin virus, or poxviruses.

Dwg.0/5

L24 ANSWER 41 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2000-452401 [39] WPIDS
CROSS REFERENCE: 2000-452400 [39]; 2000-465745 [39]

DOC. NO. CPI: C2000-137950
TITLE: **Polynucleotide** encoding antigenic type C HIV Gag polypeptide or a HIV Env polypeptide and the polypeptide useful for immunizing a mammal especially **human** against HIV.
DERWENT CLASS: B04 D16
INVENTOR(S): BARNETT, S; ZUR MEGEDE, J
PATENT ASSIGNEE(S): (CHIR) CHIRON CORP
COUNTRY COUNT: 87
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000039304	A2	20000706	(200039)*	EN	113

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG UZ VN YU ZA ZW

AU 2000024873	A	20000731	(200050)	
EP 1141314	A2	20011010	(200167)	EN

R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000039304	A2	WO 1999-US31273	19991230
AU 2000024873	A	AU 2000-24873	19991230
EP 1141314	A2	EP 1999-968202	19991230
		WO 1999-US31273	19991230

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000024873	A	Based on	WO 200039304
EP 1141314	A2	Based on	WO 200039304

PRIORITY APPLN. INFO: US 1999-152195P 19990901; US 1998-114495P 19981231
AN 2000-452401 [39] WPIDS
CR 2000-452400 [39]; 2000-465745 [39]
AB WO 200039304 A UPAB: 20011119
NOVELTY - An expression cassette (I) comprising a **polynucleotide** encoding antigenic type C HIV Gag polypeptide (IIa) or a HIV Env polypeptide (IIb), is new.

DETAILED DESCRIPTION - (I) comprises:

(a) a **polynucleotide** (Ia) encoding (IIa) having a nucleotide sequence 90% identical to a sequence having nucleotides 844-903 of a sequence (s1) having 60 bp or 841-900 of a sequence (s2) having 60 bp or a sequence (s3) having 1479 or 1509 bp as given in the specification; or

(b) a **polynucleotide** (Ib) encoding (IIb) having a nucleotide sequence 90% identical to a sequence having nucleotides 1213-1353 of 141 bp as given in the specification.

INDEPENDENT CLAIMS are also included for the following:

(1) a **recombinant** expression system (III) comprising (I)

operably linked to control elements compatible with expression in the selected host cell;

(2) a cell (IV) comprising (I) operably linked to control elements compatible with expression in the selected host cell; and

(3) a composition comprising (I) for generating an immune response.

ACTIVITY - Anti-HIV.

MECHANISM OF ACTION - Vaccine.

The plasmid DNA pCMVKM2 carrying the synthetic Gag expression cassette, was diluted to the following final concentrations in a total injection volume of 100 mu l: 20 mu g, 2 mu g, 0.2 mu g, 0.02 and 0.002 mu g. As a control, plasmid DNA of the native Gag expression cassette was handled in the same manner. Twelve groups of four or ten Balb/c mice were intramuscularly immunized for a period of 0-4 weeks. The **human** immune response was checked with an anti-HIV Gag or Env antibody ELISAs of the mice sera 0 and 4 weeks post immunization. Synthetic expression cassettes will provide a clear improvement of immunogenicity relative to the native expression cassettes. The frequency of specific cytotoxic T-lymphocytes (CTL) was evaluated by a standard chromium release assay of peptide pulsed Balb/c mouse CD4 cells. Env expressing vaccinia virus infected CD-8 cells were used as a positive control. Cytotoxic activity was measured in a standard 51Cr release assay. Target (T) cells were cultured with effector (E) cells at various E:T ratios for 4 hours and the average cpm from duplicate wells were used to calculate percent specific 51Cr release. Cytotoxic T-cell (CTL) activity was measured in splenocytes recovered from mice immunized with HIV Gag or Env DNA. Specific lysis of Gag or Env peptide-pulsed SV-BALB (MHC matched) targets cells, indicative of a CTL response. Target cells that were peptide-pulsed and derived from an MHC-unmatched mouse strain (MC57) were not lysed. Thus, synthetic Env and Gag expression cassettes exhibit increased potency for induction of cytotoxic T-lymphocyte (CTL) responses by DNA immunization.

USE - Composition comprising (I) or (IIa) or (IIb) is useful (as a vaccine) for immunizing a subject preferably a mammal especially **human** against HIV. The method comprises expressing (IV) under suitable conditions of expression, isolating (II) produced in administering it to elicit an immune response (claimed). Gag of HIV-1 self assemble into non-infectious virus-like particles which are used as a matrix for the proper presentation of an antigen entrapped or associated to the immune system of the host.

Dwg.0/6

L24 ANSWER 42 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2000-452400 [39] WPIDS
CROSS REFERENCE: 2000-452401 [39]; 2000-465745 [40]
DOC. NO. CPI: C2000-137949
TITLE: Expression cassettes encoding the **human** immunodeficiency virus (HIV) Gag-containing polypeptide useful for vaccinating against HIV infections and acquired immunodeficiency syndrome (AIDS).
DERWENT CLASS: B04 C06 D16
INVENTOR(S): BARNETT, S; GREER, C; HARTOG, K; LIAN, Y; LIU, H; SELBY, M; SRIVASTAVA, I; WALKER, C; ZUR MEGEDE, J; ZUR, M J
PATENT ASSIGNEE(S): (CHIR) CHIRON CORP
COUNTRY COUNT: 90
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000039302	A2	20000706	(200039)*	EN	390

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW

W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES
FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS
LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL
TJ TM TR TT TZ UA UG UZ VN YU ZA ZW

AU 2000022216 A 20000731 (200050)
EP 1141313 A2 20011010 (200167) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT
RO SE SI
ZA 2001005590 A 20020731 (200271) 135
JP 2002533124 W 20021008 (200281) 386
ZA 2001005589 A 20021030 (200282) 411

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000039302	A2	WO 1999-US31245	19991230
AU 2000022216	A	AU 2000-22216	19991230
EP 1141313	A2	EP 1999-966727	19991230
		WO 1999-US31245	19991230
ZA 2001005590	A	ZA 2001-5590	20010706
JP 2002533124	W	WO 1999-US31245	19991230
		JP 2000-591193	19991230
ZA 2001005589	A	ZA 2001-5589	20010706

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000022216	A	Based on	WO 200039302
EP 1141313	A2	Based on	WO 200039302
JP 2002533124	W	Based on	WO 200039302

PRIORITY APPLN. INFO: US 1999-168471P 19991201; US 1998-114495P 19981231

AN 2000-452400 [39] WPIDS

CR 2000-452401 [39]; 2000-465745 [40]

AB WO 200039302 A UPAB: 20021220

NOVELTY - Synthetic expression cassettes comprising nucleic acids encoding the **human** immunodeficiency virus (HIV) Gag-containing polypeptide, are new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) an expression cassette (I) comprising a **polynucleotide** sequence encoding a protein comprising a **human** immunodeficiency virus (HIV) Gag polypeptide (the **polynucleotide** sequence encoding the Gag polypeptide comprises a sequence with at least 90% sequence identity to a defined 60 nucleotide sequence (N1) given in the specification);

(2) a **recombinant** expression system (II) for use in a host cell comprising (I) operably linked to control elements suitable or protein expression in the host;

(3) a cell (III) comprising (II);

(4) a method (IV) for producing polypeptides including HIV Gag polypeptide sequences, comprising incubating (III) under conditions suitable for expression of the polypeptide;

(5) a method (V) for producing virus-like particles (VLPs), comprising incubating (III) under conditions suitable for production of VLPs; and

(6) a method (VI) for DNA vaccination of a subject, comprising

introducing (II) into a subject under conditions suitable for gene expression.

ACTIVITY - Anti-HIV.

MECHANISM OF ACTION - Vaccine.

USE - The expression cassettes may be used for the **recombinant** expression of HIV Gag-polypeptides which may then be used to vaccinate against HIV infection and acquired immunodeficiency syndrome (AIDS).

Dwg.0/82

L24 ANSWER 43 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2000-376316 [32] WPIDS
DOC. NO. CPI: C2000-113751
TITLE: Vaccine useful for immunizing susceptible ungulates against papillomatous digital dermatitis comprises a biologically pure culture of a Spirochete of Treponema genus.
DERWENT CLASS: B04 C06 D16
INVENTOR(S): BERRY, S L; CULLOR, J S; HIRD, D W; LEFEBVRE, R B; LEFLER, H M; READ, D H; WALKER, R L
PATENT ASSIGNEE(S): (REGC) UNIV CALIFORNIA
COUNTRY COUNT: 91
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000027429	A2	20000518	(200032)*	EN	67
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
US 6096323	A	20000801	(200039)		
AU 2000016146	A	20000529	(200041)		
EP 1128843	A2	20010905	(200151)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
US 6287575	B1	20010911	(200154)#		
JP 2002529063	W	20020910	(200274)		88

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 2000027429	A2		WO 1999-US26537	19991109
US 6096323	A	CIP of	US 1997-943571	19971003
			US 1998-191099	19981112
AU 2000016146	A		AU 2000-16146	19991109
EP 1128843	A2		EP 1999-958866	19991109
			WO 1999-US26537	19991109
US 6287575	B1		US 1997-943571	19971003
JP 2002529063	W		WO 1999-US26537	19991109
			JP 2000-580658	19991109

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000016146	A	Based on	WO 200027429

EP 1128843 A2 Based on WO 200027429
JP 2002529063 W Based on WO 200027429

PRIORITY APPLN. INFO: US 1998-191099 19981112; US 1997-943571 19971003
AN 2000-376316 [32] WPIDS
AB WO 200027429 A UPAB: 20000706
NOVELTY - A biologically pure culture (I) of ungulate Treponema, for immunizing against papillomatous digital dermatitis, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:
(1) a pharmaceutical composition comprising ungulate Treponema antigen (II); and
(2) detecting the presence of (I) nucleic acids in a biological sample comprises;
(i) contacting the sample with an oligonucleotide probe which specifically hybridizes with a Treponema-specific 16S rRNA **polynucleotide** having a fully defined 1413, 1417, 1462, 1366, 1405, 1414, 1400 or 1446 bp sequence (given in the specification), thereby forming a hybridization complex; and
(ii) detecting the presence or absence of the complex.
ACTIVITY - Antibacterial. No supporting data is given.
MECHANISM OF ACTION - None given.
USE - (II) is useful for immunizing against ungulate Treponema that is as PDD vaccines. (II) is also useful for detecting the presence of antibodies specific (II) in a biological sample preferably bovine serum, by contacting the sample with (II) immobilized on to a solid surface and detecting the presence of complex using a labeled anti-bovine antibody (claimed). An antibody preferably a monoclonal antibody specific to (II) is useful for detecting in the presence of ungulate Treponema in a sample by contacting the sample with the antibody immobilized on to a solid surface and detecting the presence of antigen/antibody complex by using a second labeled antibody. The detection method further comprises amplifying the target Treponema-specific **polynucleotide** (claimed).
Dwg.1/1

L24 ANSWER 44 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2000-293015 [25] WPIDS
DOC. NO. CPI: C2000-088548
TITLE: New **mutant** cholera holotoxin having a point mutation at amino acid position 29 of the A subunit useful as an adjuvant in an antigenic composition to enhance the immune response in a vertebrate host to a selected antigen from a pathogen.
DERWENT CLASS: B04 C06 D16
INVENTOR(S): ELDRIDGE, J H; GREEN, B A; HANCOCK, G E; HOLMES, R K; JOBLING, M G; PEEK, J A
PATENT ASSIGNEE(S): (AMCY) AMERICAN CYANAMID CO; (USSH) US DEPT HEALTH & HUMAN SERVICES; (USGO) UNIV UNIFORMED SERVICES HEALTH SCI
COUNTRY COUNT: 86
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000018434	A1	20000406	(200025)*	EN	152

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA

UG US UZ VN YU ZA ZW
AU 9964039 A 20000417 (200035)
BR 9914160 A 20010626 (200140)
EP 1117435 A1 20010725 (200143) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI
CN 1320043 A 20011031 (200215)
KR 2001085859 A 20010907 (200218)
JP 2002525093 W 20020813 (200267) 140

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000018434	A1	WO 1999-US22520	19990930
AU 9964039	A	AU 1999-64039	19990930
BR 9914160	A	BR 1999-14160	19990930
		WO 1999-US22520	19990930
EP 1117435	A1	EP 1999-951639	19990930
		WO 1999-US22520	19990930
CN 1320043	A	CN 1999-811557	19990930
KR 2001085859	A	KR 2001-703968	20010328
JP 2002525093	W	WO 1999-US22520	19990930
		JP 2000-571951	19990930

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9964039	A	Based on	WO 200018434
BR 9914160	A	Based on	WO 200018434
EP 1117435	A1	Based on	WO 200018434
JP 2002525093	W	Based on	WO 200018434

PRIORITY APPLN. INFO: US 1998-102430P 19980930
AN 2000-293015 [25] WPIDS
AB WO 200018434 A UPAB: 20000524
NOVELTY - An antigenic composition which comprises a **mutant** cholera holotoxin featuring a point mutation at amino acid 29 of the A subunit where the glutamic acid residue is replaced by an amino acid other than aspartic acid.

DETAILED DESCRIPTION - The antigenic composition (AC) enhances the immune response in a vertebrate host to an antigen selected from a pathogenic bacterium, virus, fungus or parasite. The holotoxin has reduced toxicity compared to a wild-type cholera holotoxin. INDEPENDENT CLAIMS are also included for the following:

(1) a plasmid containing an isolated and purified DNA sequence comprising a DNA sequence which encodes an immunogenic **mutant** cholera holotoxin having a substitution other than aspartic acid for the glutamic acid at position 29 of the A subunit of the cholera holotoxin and where the DNA sequence is operatively linked to an arabinose inducible promoter;

(2) a host cell transformed, transduced or transfected with the plasmid of claim (1); and

(3) producing an immunogenic **mutant** cholera holotoxin where the holotoxin has reduced toxicity compared to the wild type and has a substitution other than aspartic acid for the glutamic acid at position 29 of the A subunit of cholera holotoxin. The method comprises transforming, transducing or transfecting a host cell with the plasmid of claim (1) and culturing the host cell under conditions which permit the expression of

the **recombinant** immunogenic detoxified protein by the host cell.

ACTIVITY - Immunostimulatory. 1 micro g of CT-CRM-E29H facilitated the greatest systemic and local humoral immune responses against rP4 protein. This example describes the immune responses of BALB/c mice immunized with **recombinant** (r) P4 and P6 Outer Membrane Proteins of Nontypable Haemophilus influenzea (NTHi). In a first experiment, five BALB/c mice per group were immunized intranasally on days 0, 21 and 35 with a 10 mu l dose containing 5 micro g rP4 or 10 micro g rP6 plus 1 micro g of the adjuvant (CT, CT-B, E29H, E110D, E112D, R7K and R11K). The anti-rP4 IgG antibody titers were determined by ELISA on pooled samples collected at days 0, 21, 35 and 48. For the cholera **mutant** adjuvant E29H the titre increased from 1.052 at day 0 to 95,922 at day 48 this compared to 1,157 at day 0 to 1,968 at day 48 where no adjuvant was added.

MECHANISM OF ACTION - Induction of IgA in mucosal surfaces. The IgA response in a bronchoalveolar wash on day 49 after immunization with a dose containing rP4 and the adjuvant E29H showed titre of 845 compared to 27 when no adjuvant was added.

USE - A method is claimed for increasing the ability of an antigenic composition (AC) to enhance an immune response of a vertebrate host against a selected antigen such as a pathogenic bacterium, virus, fungus or parasite, by administration of the antigenic composition. An effective amount of the cholera holotoxin is used to enhance this immune response in a vertebrate host to the antigen. The preferred antigenic compositions listed under preferred composition are able to elicit an increased immune response of a vertebrate host. Desirable bacterial vaccines including the CT-CRM **mutants** as an adjuvant include those directed to the prevention and/or treatment of disease caused by Haemophilus influenzae, Haemophilus somnus, Moraxella catarrhalis, Streptococcus pyrogens, Streptococcus agalactiae, Helicobacter pylori, Neisseria meningitidis, Neisseria gonorrohoea Chlamydia trachomatis, Salmonella typhi, Eschericia coli, Shigella, Vibrio cholerae, Corynebacterium diphtheriae, Mycobacterium tuberculosis Mycobacterium avium-Mycobacterium intracellulare complex, Proteus mirabilis, Proteus vulgaris, Staphylococcus aureus, Clostridium tetani, Leptospira interrogans and Mycoplasma gallisepticum. Desirable viral vaccines including the CT-CRM **mutants** as an adjuvant include those directed to the prevention and/or treatment of disease caused by the following viruses: Respiratory synctial virus, **Parainfluenza** virus types 1-3, Influenza virus, Herpes simplex virus, **Human** cytometagalovirus, **Human** immunodeficiency virus, Hepatitis A, B and C, **Human** papillomavirus, poliovirus, rotavirus, calciviruses, Measles virus, Mumps virus, Rubella virus, adenovirus, rabies virus, canine distemper virus, feline leukemia virus, Marek's disease virus, equine arteritis virus and various Encephalitis viruses. Desirable vaccines against fungal pathogens include those directed to the prevention and/or treatment of disease caused by Aspergillus Blastomyces, Candida, Coccidiodes, Cryptococcus and Histoplasma. Desirable vaccines against parasites including the CR-CRM **mutants** as an adjuvant include those directed to the prevention and/or treatment of disease caused by Leishmania major, Ascaris, Trichuris, Giardia, Schistosoma, Cryptosporidium, Trichomonas, Toxoplasma gondii and Pneumocystis carinii.

ADVANTAGE - Parenteral immunization regimens are usually ineffective in inducing secretory IgA responses. However, in this approach the coadministration of (cholera toxin) CT, which is a mucosal adjuvant, with an unrelated antigen results in the induction of concurrent circulating and mucosal antibody responses to that antigen. The mutated CT has reduced toxicity so that the symptoms of diarrhoea caused by wild type CT are reduced.

Dwg.0/14

L24 ANSWER 45 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2000-182917 [16] WPIDS
DOC. NO. CPI: C2000-057410
TITLE: Inducing protective immunity against an autoimmune disease such as multiple sclerosis in mammals by vaccinating with DNA encoding a cytokine, especially a C-C family chemokine or tumor necrosis factor alpha.
DERWENT CLASS: B04 C06 D16
INVENTOR(S): KARIN, N; WILDBAUM, G; YOUSSEF, S
PATENT ASSIGNEE(S): (TECR) TECHNION RES & DEV FOUND LTD; (FRIE-I) FRIEDMAN M M; (KARI-I) KARIN N; (WILD-I) WILDBAUM G; (YOUS-I) YOUSSEF S
COUNTRY COUNT: 87
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000006203	A1	20000210	(200016)*	EN	81
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ UG ZW					
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZA ZW					
AU 9952135	A	20000221	(200029)		
EP 1100546	A1	20010523	(200130)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
US 6316420	B1	20011113	(200173)		
US 2002086841	A1	20020704	(200247)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 2000006203	A1		WO 1999-US16000	19990715
AU 9952135	A		AU 1999-52135	19990715
EP 1100546	A1		EP 1999-937261	19990715
			WO 1999-US16000	19990715
US 6316420	B1		US 1998-123485	19980728
US 2002086841	A1	Cont of	US 1998-123485	19980728
			US 2001-887031	20010625

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9952135	A	Based on	WO 200006203
EP 1100546	A1	Based on	WO 200006203
US 2002086841	A1	Cont of	US 6316420

PRIORITY APPLN. INFO: US 1998-123485 19980728; US 2001-887031 20010625
AN 2000-182917 [16] WPIDS
AB WO 200006203 A UPAB: 20000330
NOVELTY - Inducing protective immunity against an autoimmune disease in a mammal by vaccinating with DNA construct (I) encoding a cytokine, is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) inducing protective immunity against an autoimmune disease comprising removing cells from a mammal, transducing the cells with (I) and reintroducing the cells to the mammal.

(2) a pharmaceutical composition comprising a construct as above and a carrier; and

(3) antibodies against a cytokine expressed by cells transduced with a construct which includes a **polynucleotide** encoding the cytokine operatively linked to one or more transcription control sequences.

ACTIVITY - Immunosuppressive; neuroprotectant.

MECHANISM OF ACTION - Vaccine.

USE - The methods and composition are useful to induce protective immunity against autoimmune diseases, especially multiple sclerosis (claimed) in mammals e.g. dogs, cattle pigs and especially **humans** (claimed).

ADVANTAGE - DNA vaccination resulted in the generation of immunity to autologous antigens, overcoming the problems of immunogenicity associated with treating chronic diseases using xenogenic neutralizing antibodies. The immune response to the DNA constructs also appeared to be elicited only during the course of the disease in studies of experimental autoimmune encephalomyelitis (EAE; an autoimmune disease of the central nervous system which serves as a model for multiple sclerosis), advantageous to restrain the potentially harmful reactivity of the immune system to times when such a response is needed in diseases caused by a malfunction in the immune system in distinguishing self from foreign.

Dwg.0/12

L24 ANSWER 46 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2000-108401 [10] WPIDS
CROSS REFERENCE: 2002-354207 [39]
DOC. NO. CPI: C2000-032768
TITLE: Novel vector used for gene transfer methods, e.g. gene therapy.
DERWENT CLASS: B04 D16
INVENTOR(S): LUSKY, M; MEHTALI, M
PATENT ASSIGNEE(S): (TRGE) TRANSGENE SA
COUNTRY COUNT: 29
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
EP 974668	A1	20000126	(200010)*	EN	23
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
CA 2276791	A1	20000107	(200025)	EN	
JP 2000106875	A	20000418	(200030)		55
AU 9938009	A	20000608	(200035)		
EP 974668	B1	20021002	(200272)	EN	
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					
US 6475480	B1	20021105	(200276)		
DE 69903229	E	20021107	(200281)		
ES 2180258	T3	20030201	(200322)		
AU 756607	B	20030116	(200324)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
EP 974668	A1	EP 1999-401645	19990701
CA 2276791	A1	CA 1999-2276791	19990706

JP 2000106875	A		JP 1999-227880	19990707
AU 9938009	A		AU 1999-38009	19990706
EP 974668	B1		EP 1999-401645	19990701
		Related to	EP 2001-124236	19990701
US 6475480	B1		US 1999-348049	19990706
DE 69903229	E		DE 1999-603229	19990701
			EP 1999-401645	19990701
ES 2180258	T3		EP 1999-401645	19990701
AU 756607	B		AU 1999-38009	19990706

FILING DETAILS:

PATENT NO	KIND		PATENT NO
EP 974668	B1	Related to	EP 1199368
DE 69903229	E	Based on	EP 974668
ES 2180258	T3	Based on	EP 974668
AU 756607	B	Previous Publ.	AU 9938009

PRIORITY APPLN. INFO: EP 1998-402825 19981113; EP 1998-401722 19980707

AN 2000-108401 [10] WPIDS

CR 2002-354207 [39]

AB EP 974668 A UPAB: 20030410

NOVELTY - A **recombinant** adenoviral vector derived from an adenovirus genome, where at least a part of E1 region is deleted or nonfunctional and a part of E4 region is deleted, and comprising a gene of interest operably linked to regulatory elements, each vector retains sufficient E4 sequences to improve expression and/or persistence of expression of the gene of interest in a host cell or organism, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) use of a **polynucleotide** comprising one or more open reading frames (ORF)s of the E4 region of an adenovirus selected from the group consisting of ORF1,2,3,4,3/4,6/7,6 and 7, taken individually or in combination, to improve the expression and/or persistence of expression of a gene of interest, operably linked to regulatory elements and inserted into an expression vector;

(2) a non-adenoviral vector comprising a gene of interest operably linked to regulatory elements and comprising the **polynucleotide** of (1);

(3) an infectious viral particle comprising the novel vector or an expression vector containing the E4 ORFs;

(4) a method of preparing an infectious viral particle of (3), comprising introducing the novel vector or an expression vector, containing the E4 ORFs into a complementation cell capable of complementing in trans the vector, culturing the transfected complementation cell to produce the infectious viral particle and recovering it from the cell culture;

(5) a host cell comprising the novel vector, the expression vector containing the E4 ORFs, or infected by the infectious viral particle of (3);

(6) a composition comprising the novel vector, the expression vector containing the E4 ORFs, the infectious viral particles or the host cells of (5);

(7) use of the novel vector, the expression vector containing the ORFs, the infectious viral particles or the host cells, to form a pharmaceutical composition, intended for gene transfer; and

(8) a product comprising an expression vector comprising a gene of interest operably linked to regulatory elements, and a

polynucleotide of (1), as a combination product for simultaneous or separate use.

ACTIVITY - None given

MECHANISM OF ACTION - The vectors can be used for gene therapy.

USE - The novel vector is used in a therapeutic composition, for gene transfer (claimed).

ADVANTAGE - The expression vectors do not show the instability of transgene expression observed in E4-deleted adenovirus vectors, due to the retention of the selected E4 ORF sequences, allowing long term expression of the transgene in the cells

Dwg.0/0

L24 ANSWER 47 OF 67 MEDLINE DUPLICATE 4

ACCESSION NUMBER: 2001046569 MEDLINE

DOCUMENT NUMBER: 20523897 PubMed ID: 11069997

TITLE: **Respiratory syncytial virus** can tolerate an intergenic sequence of at least 160 nucleotides with little effect on transcription or replication in vitro and in vivo.

AUTHOR: Bukreyev A; Murphy B R; Collins P L

CORPORATE SOURCE: Laboratory of Infectious Diseases, National Institute of Allergy and Infectious Diseases, Bethesda, Maryland 20892-0720, USA.

SOURCE: JOURNAL OF VIROLOGY, (2000 Dec) 74 (23) 11017-26. Journal code: 0113724. ISSN: 0022-538X.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200012

ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001204

AB The intergenic sequences (IGS) between the first nine genes of **human respiratory syncytial virus** (**RSV**) vary in length from 1 to 56 nucleotides and lack apparent conserved sequence motifs. To investigate their influence on sequential transcription and viral growth, **recombinant RSV** strain A2, from which the **SH** gene had been **deleted** to facilitate manipulation, was further modified to contain an M-G IGS of 16, 30, 44, 58, 65, 72, 86, 100, 120, 140, or 160 nucleotides. All of the viruses were viable. For viruses with an M-G IGS of 100 nucleotides or more, plaque size decreased with increasing IGS length. In this same length range, increasing IGS length was associated with modest attenuation during single-step, but not multistep, growth in HEp-2 cells. Surprisingly, Northern blot analysis of the accumulation of six different mRNAs indicated that there was little or no change in transcription with increasing IGS length. Thus, the **RSV** polymerase apparently can readily cross IGS of various lengths, including unnaturally long ones, with little or no effect on the efficiency of termination and reinitiation. This finding supports the view that the IGS do not have much effect on sequential transcription and provides evidence from infectious virus that IGS length is not an important regulatory feature. To evaluate replication in vivo, BALB/c mice were infected intranasally with **RSV** containing an M-G IGS of 65, 140, or 160 nucleotides. Replication of the latter two viruses was decreased up to 5- and 25-fold in the upper and lower respiratory tracts, respectively, on day 3 following infection. However, the level of replication at both sites on days 4 and 5 was very similar to that of the virus with an IGS of 65 nucleotides. Thus, the modest attenuation in vivo associated with the

longer IGS was additive to that conferred by **deletion** of the **SH** gene and might be useful to incrementally increase the level of attenuation of a live-attenuated vaccine virus.

L24 ANSWER 48 OF 67 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 2000319123 MEDLINE
DOCUMENT NUMBER: 20319123 PubMed ID: 10860892
TITLE: Role of type I IFNs in the in vitro attenuation of live, temperature-sensitive vaccine strains of **human respiratory syncytial virus**.
AUTHOR: Loveys D A; Kulkarni S; Atreya P L
CORPORATE SOURCE: Laboratory of Pediatric and Respiratory Viral Diseases, DVP/CBER, Food and Drug Administration, Bethesda, MD 20892, USA.
SOURCE: VIROLOGY, (2000 Jun 5) 271 (2) 390-400.
Journal code: 0110674. ISSN: 0042-6822.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 20000811
Last Updated on STN: 20000811
Entered Medline: 20000802

AB The contributions of type I interferons (IFNs) to the in vitro attenuation of three temperature-sensitive (Ts) subgroup A and one subgroup B deletion **mutant RSV** strains were evaluated. The ability of these vaccine viruses to induce IFNs at their permissive and restrictive temperatures and their sensitivity to the antiviral effects of exogenous I IFNs were tested in **human** lung epithelial A549 cells. Our results show that the highly attenuated and immunogenic subgroup A vaccine strain Ts1C produced higher levels of IFN-beta than its parent RSS-2 or two related strains, Ts1A and Ts1B, at their permissive temperature. Growth of **RSV**-infected A549 cultures at restrictive temperatures or prior UV inactivation of the virus abolished the observed induction of IFN-beta, suggesting a strict requirement of viral replication for cellular IFN induction. The enhanced induction of IFN-beta by the highly immunogenic Ts1C at permissive temperature may be an advantageous characteristic of a live intranasal vaccine candidate. The subgroup B strain **RSV** B1 and its **mutant** cp-52 (with **SH** and G gene **deletions**) both induced similar but low levels of IFN-beta. Hence the observed overattenuation of cp-52 in **human** infants is probably not due to enhanced IFN induction during its replication in the host. The ability of cp-52, which does not express the SH and G proteins, to induce IFN-beta levels similar to those of its parent strain suggests that these viral proteins may not have a role in the induction of IFN-beta in the host. In addition, both subgroup A and B **mutants** and their respective parent strains were similarly resistant to the antiviral effects of exogenous IFN-alpha or -beta. Therefore, increased sensitivity of the **mutants** to IFNs does not seem to contribute to their attenuation.

L24 ANSWER 49 OF 67 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 2000399358 MEDLINE
DOCUMENT NUMBER: 20351739 PubMed ID: 10891423
TITLE: **Recombinant respiratory syncytial viruses** with **deletions** in the NS1, NS2, **SH**, and M2-2 genes are attenuated in vitro and in vivo.

AUTHOR: Jin H; Zhou H; Cheng X; Tang R; Munoz M; Nguyen N
CORPORATE SOURCE: Aviron, 297 North Bernardo Avenue, Mountain View, California, 94043, USA.. hjin@aviron.com
SOURCE: VIROLOGY, (2000 Jul 20) 273 (1) 210-8.
Journal code: 0110674. ISSN: 0042-6822.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 20000824
Last Updated on STN: 20000824
Entered Medline: 20000811

AB **Respiratory syncytial virus** (**RSV**) encodes several proteins that lack well-defined functions; these include NS1, NS2, SH, and M2-2. Previous work has demonstrated that NS2, SH, and M2-2 can each be deleted from **RSV** genome and thus are considered as accessory proteins. To determine whether **RSV** can replicate efficiently when two or more transcriptional units are **deleted**, we removed NS1, NS2, **SH**, and M2-2 genes individually and in different combinations from an infectious cDNA clone derived from **human RSV** A2 strain. The following six **mutants** with two or more genes deleted were obtained: DeltaNS1NS2, DeltaM2-2SH, DeltaM2-2NS2, DeltaSHNS1, DeltaSHNS2, and DeltaSHNS1NS2. Deletion of M2-2 together with NS1 was detrimental to **RSV** replication. It was not possible to obtain a **recombinant RSV** when all four genes were deleted. All of the double and triple deletion **mutants** exhibited reduced replication and small plaque morphology in vitro. Replication of these deletion **mutants** was more reduced in HEp-2 cells than in Vero cells. Among the 10 single and multiple gene deletion **mutants** obtained, DeltaM2-2NS2 was most attenuated. DeltaM2-2NS2 formed barely visible plaques in HEp-2 cells and had a reduction of titer of 3 log(10) compared with the wild-type **recombinant RSV** in infected HEp-2 cells. When inoculated intranasally into cotton rats, all of the deletion **mutants** were attenuated in the respiratory tract. Our data indicated that the NS1, NS2, SH, and M2-2 proteins, although dispensable for virus replication in vitro, provide auxiliary functions for efficient **RSV** replication.

L24 ANSWER 50 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2000-086973 [07] WPIDS
DOC. NO. CPI: C2000-024257
TITLE: Novel heat shock procedure and **recombinant** viruses useful for diagnostic research studies and as therapeutic or prophylactic vaccines.
DERWENT CLASS: B04 C06 D16
INVENTOR(S): KOVACS, G R; PARKS, C L; SIDHU, M S; UDEM, S A
PATENT ASSIGNEE(S): (AMCY) AMERICAN CYANAMID CO
COUNTRY COUNT: 85
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9963064	A1	19991209	(200007)*	EN	75

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA

UG US UZ VN YU ZA ZW
AU 9944144 A 19991220 (200021)
BR 9910929 A 20010220 (200114)
EP 1090108 A1 20010411 (200121) EN
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU NL PT SE
CN 1303426 A 20010711 (200159)
MX 2000011420 A1 20010401 (200171)
KR 2001052498 A 20010625 (200173)
JP 2002517189 W 20020618 (200242) 74

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9963064	A1	WO 1999-US12292	19990603
AU 9944144	A	AU 1999-44144	19990603
BR 9910929	A	BR 1999-10929	19990603
		WO 1999-US12292	19990603
EP 1090108	A1	EP 1999-927175	19990603
		WO 1999-US12292	19990603
CN 1303426	A	CN 1999-806717	19990603
MX 2000011420	A1	MX 2000-11420	20001121
KR 2001052498	A	KR 2000-713629	20001201
JP 2002517189	W	WO 1999-US12292	19990603
		JP 2000-552260	19990603

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9944144	A	Based on	WO 9963064
BR 9910929	A	Based on	WO 9963064
EP 1090108	A1	Based on	WO 9963064
JP 2002517189	W	Based on	WO 9963064

PRIORITY APPLN. INFO: US 1998-87800P 19980603
AN 2000-086973 [07] WPIDS
AB WO 9963064 A UPAB: 20020128
NOVELTY - A heat shock procedure for increased recovery of **recombinant** Mononegavirales virus, is new.
DETAILED DESCRIPTION - A method for producing a **recombinant** Mononegavirales virus comprises: in at least one host cell, conducting transfection, in media, of a rescue composition which comprises: a transcription vector comprising an isolated nucleic acid molecule which comprises a **polynucleotide** sequence encoding a genome or anti-genome of a non-segmented, negative-sense, single stranded RNA virus of the Mononegavirales order; and at least one expression vector which comprises one or more isolated nucleic acid molecule(s) encoding the trans-acting proteins necessary for encapsidation, transcription and replication; under conditions sufficient to permit the co-expression of the vectors and the production of the **recombinant** virus; and heating the transfected rescue composition to an effective heat shock temperature under conditions sufficient to increase the recovery of the **recombinant** virus; or transferring the transfected rescue composition onto at least one layer of plaque expansion cells.
INDEPENDENT CLAIMS are also included for the following:
(1) a **recombinant** virus prepared by a method as above; and
(2) a composition comprising a **recombinant** virus prepared as above and a pharmaceutically acceptable carrier.
ACTIVITY - Anti-viral.

MECHANISM OF ACTION - Vaccine.

USE - The **recombinant** viruses formed by the methods are useful as tools in diagnostic research studies or as therapeutic or prophylactic vaccines. The heat shock procedure can be used to improve the efficiency of the procedure used to produce virus-like particles by packaging synthetic influenza-like CAT:RNA mini-genome in the COS-1 cells, by vaccinia-T7 polymerase expressing cDNA clones of 10 influenza A virus-coded proteins. The method can also be used to improve efficiency of a helper independent system for the rescue of a segmented, negative strand RNA genome of Bunyamwera bunyavirus.

ADVANTAGE - The ability to obtain replicating virus from rescue may diminish as the **polynucleotide** encoding the native genome and anti-genome is increasingly modified. The methods of the invention improve the likelihood of **recombinant** virus rescue. An advantage of using of DNA synthesis inhibitors during a genetic rescue event is that there should be very little or no contamination of the rescued RNA virus with a modified helper virus. Heat shock temperatures above the standard temperature for performing rescue of a **recombinant** virus increase the recovery of the desired **recombinant** virus over the level of recovery of **recombinant** virus when rescue is performed in the absence of the increase in temperature.

Dwg.0/6

L24 ANSWER 51 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 2000-013259 [01] WPIDS
DOC. NO. CPI: C2000-002530
TITLE: New modified immunoglobulin molecules, useful for treatment and prevention of infectious diseases.
DERWENT CLASS: B04 C06 D16
INVENTOR(S): CHINTALACHARUVU, K R; COLOMA, M J; MORRISON, S L; TRINH, K M; YOO, E M
PATENT ASSIGNEE(S): (REGC) UNIV CALIFORNIA
COUNTRY COUNT: 86
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9954484	A1	19991028	(200001)*	EN	67
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ UG ZW					
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG UZ VN YU ZA ZW					
AU 9936558	A	19991108	(200014)		
US 6284536	B1	20010904	(200154)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 9954484	A1		WO 1999-US8647	19990420
AU 9936558	A		AU 1999-36558	19990420
US 6284536	B1	Provisional	US 1998-82578P	19980420
		Provisional	US 1998-96085P	19980811
			US 1999-295283	19990420

FILING DETAILS:

PATENT NO	KIND	PATENT NO

AU 9936558 A Based on WO 9954484

PRIORITY APPLN. INFO: US 1998-96085P 19980811; US 1998-82578P 19980420; US 1999-295283 19990420

AN 2000-013259 [01] WPIDS

AB WO 9954484 A UPAB: 20000105

NOVELTY - A modified immunoglobulin molecule (I) comprising a constant domain of an IgA molecule and at least a portion of a non IgA immunoglobulin molecule is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a **polynucleotide** (II) that encodes (I);

(2) a vector (II) that comprises (II);

(3) a host cell (IV) transfected with (III); and

(4) production of (I) by culturing (IV) and recovering (I).

ACTIVITY - Antibacterial; antiviral; antifungal; antimycoplasma; antimycobacterial; antiparasitic.

MECHANISM OF ACTION - None given.

USE - (I) may be used to treat or prevent a systemic, local or mucosal infection in a mammal, bird, reptile or fish. The types of infection that may be treated include bacterial, mycoplasmal, mycobacterial, yeast or parasitic infections, especially HIV, hepatitis, **respiratory syncytial virus**, influenza or cold viral infections (all claimed).

ADVANTAGE - The design of (I) makes it possible to overcome the limitations of naturally-occuring antibodies and desired features of one Ig class can be combines with desired features characteristic of another class.

DESCRIPTION OF DRAWING(S) - Figure A is a schematic diagram of H chain constant region genes showing where unique pVUI restriction sites were introduced between the C-H1 and C-H2 of alpha 1 and gamma 1.

Dwg.1A/14

L24 ANSWER 52 OF 67 WPIDS (C) 2003 THOMSON DERWENT

ACCESSION NUMBER: 2000-052702 [04] WPIDS

DOC. NO. CPI: C2000-013553

TITLE: DNA fragmentation factor DFF40 involved in apoptosis and related **polynucleotide**.

DERWENT CLASS: B04 D16

INVENTOR(S): LIU, X; WANG, X

PATENT ASSIGNEE(S): (TEXA) UNIV TEXAS SYSTEM

COUNTRY COUNT: 82

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9954482	A1	19991028	(200004)*	EN	154

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW

W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM GW HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW

AU 9871374 A 19991108 (200014)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE

WO 9954482	A1	WO 1998-US7895	19980416
AU 9871374	A	AU 1998-71374	19980416
		WO 1998-US7895	19980416

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9871374	A	Based on	WO 9954482

PRIORITY APPLN. INFO: WO 1998-US7895 19980416
AN 2000-052702 [04] WPIDS
AB WO 9954482 A UPAB: 20000124
NOVELTY - An isolated polypeptide encoding a DFF40 DNA fragmentation factor is new.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:
(1) an isolated peptide having 10-50 consecutive residues of DFF40;
(2) a monoclonal antibody (MAb) that binds immunologically to DFF40;
(3) a hybridoma cell that produces a MAb as in (2);
(4) a polyclonal antisera, antibodies of which bind immunologically to DFF40;
(5) an isolated nucleic acid comprising a region, or the complement, encoding DFF40 or an allelic **variant** of it;
(6) an isolated oligonucleotide of 15-50 consecutive bases of (5);
(7) a plasmid construct comprising a first nucleic acid as in (5);
(8) a method of inducing apoptosis in a cell comprises providing the cell with DFF40 which results in apoptosis;
(9) a method for inhibiting the growth of a cancer cell comprising contacting a cancer cell with a DNA fragmentation factor designated DFF40 under conditions permitting the uptake of the DNA fragmentation factor by the cell where the presence of the DFF40 into the cell induces apoptosis;
(10) a method for treating cancer comprising:
(a) encoding a DFF40 DNA fragmentation factor; and
(b) a promoter active in the tumor cell, where the promoter is operably linked to the region encoding the DNA fragmentation factor, under conditions permitting the uptake of the nucleic acid by the tumor cell;
(11) a method of identifying a modulator of DFF40;
(12) a modulator of apoptotic activity identified as in (9);
(13) an isolated DNA fragmentation factor complex for regulating chromatin stability, the complex comprising DFF40 and DFF45; and
(14) a method of producing a functional DNA fragmentation factor.
ACTIVITY - Cytostatic.
MECHANISM OF ACTION - Gene Therapy.
USE - The expression construct encoding the DFF40 and DFF45 complex is provided to a cell to induce apoptosis, especially in tumor cells. DFF40 is used to inhibit the growth of a cancer cell, especially in **humans**.
Dwg.0/1

L24 ANSWER 53 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 1999-339975 [29] WPIDS
DOC. NO. CPI: C1999-100231
TITLE: New dihydrofolate reductase-thymidylate synthase (DHFR-TS) protein and gene, useful in genetic constructs for preparing a live vaccine against Neospora.
DERWENT CLASS: B04 C06 D16
INVENTOR(S): DURTSCHI, B A; KRISHNAN, B R; YODER, S C
PATENT ASSIGNEE(S): (PFIZ) PFIZER PROD INC; (AMWA-N) AMWAY CORP; (LITT-N) LITTLE TIKES CO; (PFIZ) PFIZER INC
COUNTRY COUNT: 35

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
EP 924295	A2	19990623	(199929)*	EN	33
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
AU 9895205	A	19990624	(199936)		
CA 2253011	A1	19990604	(199947)	EN	
CN 1225945	A	19990818	(199951)		
JP 11266879	A	19991005	(199953)		30
BR 9805135	A	20000321	(200028)		
NZ 333160	A	20000623	(200038)		
ZA 9811058	A	20000726	(200042)		50
KR 2000006088	A	20000125	(200063)		
MX 9810218	A1	20000201	(200123)		
AU 738142	B	20010913	(200164)		
JP 3245398	B2	20020115	(200206)		29
US 6436410	B1	20020820	(200257)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
EP 924295	A2		EP 1998-309714	19981126
AU 9895205	A		AU 1998-95205	19981203
CA 2253011	A1		CA 1998-2253011	19981202
CN 1225945	A		CN 1998-122981	19981202
JP 11266879	A		JP 1998-345004	19981204
BR 9805135	A		BR 1998-5135	19981203
NZ 333160	A		NZ 1998-333160	19981203
ZA 9811058	A		ZA 1998-11058	19981203
KR 2000006088	A		KR 1999-21644	19990610
MX 9810218	A1		MX 1998-10218	19981203
AU 738142	B		AU 1998-95205	19981203
JP 3245398	B2		JP 1998-345004	19981204
US 6436410	B1	Provisional	US 1997-67507P	19971204
		Provisional	US 1998-95213P	19980803
			US 1998-203895	19981202

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 738142	B	Previous Publ.	AU 9895205
JP 3245398	B2	Previous Publ.	JP 11266879

PRIORITY APPLN. INFO: US 1998-95213P 19980803; US 1997-67507P 19971204; US 1998-95213 19980610; US 1998-203895 19981202

AN 1999-339975 [29] WPIDS

AB EP 924295 A UPAB: 19991122

NOVELTY - A substantially purified protein (I) comprising a fully defined 612 amino acid Neospora canium sequence given in the specification, or the amino acid sequence of a dihydrofolate reductase-thymidylate synthase (DHFR-TS) protein encoded by the DHFR-TS gene present in phage lambda NcIDHFRTS (ATCC No. 209512).

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated **polynucleotide** (II) molecule comprising a

nucleotide sequence that encodes a Neospora DHFR-TS protein;
(2) an isolated **polynucleotide** substantially homologous to (II);
(3) an isolated **polynucleotide** comprising a nucleotide sequence that encodes a polypeptide homologous to (I);
(4) a **recombinant** vector comprising the above **polynucleotide** sequences or portions of;
(5) a cell transformed with the vector;
(6) a substantially purified polypeptide (III) homologous to (I);
(7) a peptide fragment of (I) or (III);
(8) an antibody specific to (I);
(9) a genetic construct that can be used to disable a Neospora DHFR-TS gene, comprising one of the above **polynucleotides** or a portion of, further comprising at least one mutation disabling the Neospora DHFR-TS gene, or a **polynucleotide** molecule consisting of at least one nucleotide sequence that flank in situ the open reading frame of a Neospora DHFR-TS gene;
(10) a Neospora cell transformed with the genetic construct;
(11) preparation of live Neospora cells;
(12) a vaccine for protecting a mammal against neosporosis, comprising live Neospora cells that exhibit a **mutant** phenotype selected from dhfr-, ts- and dhfr-ts-.
(13) preparation of the vaccine; and
(14) a kit for vaccinating a mammal against neosporosis, comprising a first container of modified live Neospora cells (1), and a second container having a veterinary acceptable carrier or diluent.
USE - The genetic construct is useful for disrupting the DHFR domain or TS domain from the Neospora DHFR-TS gene, and the construct is useful for transforming live cells to reduce their pathogenicity to produce cells (10), which are useful as modified live vaccines for protecting a mammal against neosporosis. A combination vaccine further comprising a second component which induces a protective response against a pathogen, is useful for diseases caused by bovine herpes virus, bovine **respiratory syncytial virus**, bovine viral diarrhea virus, **parainfluenza** virus types I, II or III, Leptospira spp., Campylobacter spp., Staphylococcus aureus, Streptococcus agalactiae, Mycoplasma spp., Kleibsiella spp., Salmonella spp., rotavirus, coronavirus, rabies, Pasteurella hemolytica, Pasteurella multocida, Clostridia spp., Tetanus toxoid, E. coli, Crytosporidium spp., Eimeria spp., or Trichomonas spp. (all claimed).
The DHFR-TS proteins are useful for screening for inhibitory agents that specifically block the enzymatic activity of the DHFR or TS domain, and are also useful as antigens for raising antibodies, which are useful as diagnostic reagents to detect the presence of Neospora-specific DHFR-TS protein in a sample, or as affinity reagents for purification of DHFR-TS protein.
Dwg.0/0

L24 ANSWER 54 OF 67 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 1999174046 MEDLINE
DOCUMENT NUMBER: 99174046 PubMed ID: 10074199
TITLE: **Recombinant respiratory syncytial virus** bearing a **deletion** of either the NS2 or **SH** gene is attenuated in chimpanzees.
AUTHOR: Whitehead S S; Bukreyev A; Teng M N; Firestone C Y; St Claire M; Elkins W R; Collins P L; Murphy B R
CORPORATE SOURCE: Respiratory Viruses Section, Laboratory of Infectious Diseases, National Institute of Allergy and Infectious Diseases, Bethesda, Maryland 20892, USA..

sswhitehead@nih.gov
CONTRACT NUMBER: AI-000030 (NIAID)
AI-000087 (NIAID)
SOURCE: JOURNAL OF VIROLOGY, (1999 Apr) 73 (4) 3438-42.
Journal code: 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199905
ENTRY DATE: Entered STN: 19990517
Last Updated on STN: 19990517
Entered Medline: 19990506
AB The NS2 and SH genes of **respiratory syncytial virus** (**RSV**) have been separately deleted from a **recombinant** wild-type **RSV** strain, A2 (M. N. Teng and P. L. Collins, J. Virol. 73:466-473, 1998; A. Bukreyev et al., J. Virol. 71:8973-8982, 1997; and this study). The resulting viruses, designated rA2DeltaNS2 and rA2DeltaSH, were administered to chimpanzees to evaluate their levels of attenuation and immunogenicity. **Recombinant** virus rA2DeltaNS2 replicated to moderate levels in the upper respiratory tract, was highly attenuated in the lower respiratory tract, and induced significant resistance to challenge with wild-type **RSV**. The replication of rA2DeltaSH virus was only moderately reduced in the lower, but not the upper, respiratory tract. However, chimpanzees infected with either virus developed significantly less rhinorrhea than those infected with wild-type **RSV**. These findings demonstrate that a **recombinant RSV mutant** lacking either the NS2 or SH gene is attenuated and indicate that these deletions may be useful as attenuating mutations in new, live **recombinant RSV** vaccine candidates for both pediatric and elderly populations. The DeltaSH mutation was incorporated into a **recombinant** form of the cpts248/404 vaccine candidate, was evaluated for safety in seronegative chimpanzees, and can now be evaluated as a vaccine for **humans**.

L24 ANSWER 55 OF 67 MEDLINE
ACCESSION NUMBER: 2000015405 MEDLINE
DOCUMENT NUMBER: 20015405 PubMed ID: 10547682
TITLE: Rational design of live-attenuated **recombinant** vaccine virus for **human respiratory syncytial virus** by reverse genetics.
AUTHOR: Collins P L; Whitehead S S; Bukreyev A; Fearns R; Teng M N; Juhasz K; Chanock R M; Murphy B R
CORPORATE SOURCE: Laboratory of Infectious Diseases, National Institute of Allergy and Infectious Diseases, Bethesda, Maryland 20892-0720, USA.
SOURCE: ADVANCES IN VIRUS RESEARCH, (1999) 54 423-51. Ref: 49
Journal code: 0370441. ISSN: 0065-3527.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199912
ENTRY DATE: Entered STN: 20000113
Last Updated on STN: 20000113
Entered Medline: 19991207
AB **RSV** is a major cause of pediatric respiratory tract disease

worldwide, but a vaccine is not yet available. It is now possible to prepare live infectious **RSV** completely from cDNA. This provides a method for introducing defined mutations into infectious virus, making possible the rational design of a live-attenuated vaccine virus for intranasal administration. This is particularly important for **RSV**, for which achieving the appropriate balance between attenuation and immunogenicity by conventional methods has proven elusive. We took advantage of the existence of a panel of biologically derived vaccine candidate viruses that were incompletely attenuated but well characterized biologically. The mutations in these viruses were identified by sequence analysis and characterized by insertion into **recombinant** virus, thereby providing a menu of known attenuating mutations. These included a series of amino acid point mutations, mostly in the L polymerase, and a nucleotide substitution in a transcription gene-start signal, a cis-acting RNA element. The second source of mutations was from experimental mutational analysis of **recombinant** virus and involves **deletion** of the NS1, NS2, or **SH** gene. We have reconstructed a previously tested, biologically derived attenuated virus, cpts248/404, in **recombinant** form and are now proceeding to introduce additional mutations from the menu to achieve stepwise increases in attenuation. The ability to modify the attenuation phenotype incrementally in a directed manner should result in an appropriate vaccine virus.

L24 ANSWER 56 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 1999-081221 [07] WPIDS
DOC. NO. CPI: C1999-024467
TITLE: New nucleic acid encoding the L subunit of polymerase from paramyxoviruses - used for differential diagnosis of infections by respiratory syncytial and **parainfluenza** viruses.
DERWENT CLASS: B04 D16
INVENTOR(S): EUGENE, R G; FREYMUTH, F; TORDO, N; EUGENE-RUELLAN, G
PATENT ASSIGNEE(S): (INSP) INST PASTEUR
COUNTRY COUNT: 82
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9858959	A1	19981230	(199907)*	FR	111
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM GW HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW					
FR 2764903	A1	19981224	(199907)		
AU 9883429	A	19990104	(199921)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9858959	A1	WO 1998-FR1323	19980623
FR 2764903	A1	FR 1997-7803	19970623
AU 9883429	A	AU 1998-83429	19980623

FILING DETAILS:

PATENT NO	KIND	PATENT NO

AU 9883429 A Based on WO 9858959

PRIORITY APPLN. INFO: FR 1997-7803 19970623
AN 1999-081221 [07] WPIDS
AB WO 9858959 A UPAB: 19990224
New **polynucleotide** (I): (a) contains at least 12 consecutive nucleotides (nt) from a 355 bp sequence (1), given in the specification; (b) is complementary to (a); or (c) hybridises under highly stringent conditions with (a) or (b). Also new are: (A) primers, and their pairs, for specific amplification of (1); (B) polypeptides (II), corresponding to region III of the large subunit (L) of the polymerase gene of **RSV** (**respiratory syncytial virus**)B, that is (a) the sequence TTDLSKFNQAFRYETSCICSDVLDELHGVQSLFSWLHLTIPLVTIICTYRHAPPFIKDHVV NLNEVDEQSGLYRYHMGGIEGWCQKLWTIEAISLLDLISLKGKFSITALINGDNQAI (6), (b) a fragment of at least 10 amino acids (aa) from (6) or (c) a modified form of (a) or (b) with at least one substitution, insertion or deletion, with (b) and (c) still able to bind antibodies against region III; (C) any **polynucleotide** (Ia) that encodes (II); (D) **recombinant** vector (for cloning, expression or insertion) containing (I) or (Ia); (E) **recombinant** host cells containing (I), (Ia) or the vector; (F) antibodies (Ab), mono- or poly-clonal, specific for (II); (G) **polynucleotide** (Ib), of at least 12 nt, able to hybridise specifically under highly stringent conditions to a nucleic acid encoding L of a specific virus of the family Paramyxoviridae, or to its complement; (H) primers (and their pairs) for amplifying genomic DNA or cDNA of **RSV** or **parainfluenza** virus (PIV) corresponding to specific regions of the L gene; (J) **polynucleotides** (Ic) able to hybridise under highly stringent conditions with nucleic acid encoding the L gene subunit of **RSV** A or B, and PIV3.
USE - (I), (Ia) and their fragments are primers and probes for specific detection of **RSV** A or B and PIV3, in usual amplification or hybridisation assays. Ab are used to detect the same viruses at the protein level (in standard immunoassays) and (II) are used to detect virus-specific antibodies similarly. Cells of (E) are used to produce **recombinant** (II), useful as immunogens.
ADVANTAGE - These nucleic acids allow early differential diagnosis of infection by **RSV** and PIV3 (for optimisation of antiviral therapy and to prevent nocosomal transfer).
Dwg.0/7

L24 ANSWER 57 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 1999-080950 [07] WPIDS
DOC. NO. CPI: C1999-024320
TITLE: Producing secretory immunoglobulin in single cells - useful to produce commercial quantities of secretory immunoglobulin to prevent or treat infections.
DERWENT CLASS: B04 C06 D16
INVENTOR(S): CHINTALACHARUVU, K R; MORRISON, S L
PATENT ASSIGNEE(S): (REGC) UNIV CALIFORNIA
COUNTRY COUNT: 82
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9857993	A1	19981223	(199907)*	EN	39

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM GW HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG

MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG
UZ VN YU ZW
AU 9880637 A 19990104 (199921)
US 6300104 B1 20011009 (200162)
US 2002127645 A1 20020912 (200262)

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 9857993	A1		WO 1998-US11975	19980610
AU 9880637	A		AU 1998-80637	19980610
US 6300104	B1	Provisional	US 1997-50969P	19970619
			US 1998-95385	19980610
US 2002127645	A1	Provisional Div ex	US 1997-50969P	19970619
			US 1998-95385	19980610
			US 2001-950294	20010910

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9880637	A	Based on	WO 9857993
US 2002127645	A1	Div ex	US 6300104

PRIORITY APPLN. INFO: US 1997-50969P 19970619; US 1998-95385
19980610; US 2001-950294 20010910
AN 1999-080950 [07] WPIDS
AB WO 9857993 A UPAB: 19990217
A novel method of producing secretory immunoglobulin (sIg) molecules comprises transfecting a cell producing an immunoglobulin (Ig) with a **polynucleotide** encoding a secretory component (SC) to form SC transfected Ig producing cells. Also claimed is a secretory immunoglobulinA (sIgA) produced as above.

USE - The method is useful to produce commercial quantities of sIg (especially sIgA) to treat or prevent infections. In particular, sIgA produced by the method can be combined with a carrier in pharmaceutical compositions (claimed), which can be administered to prevent/treat infections (claimed) especially in mammals (particularly **humans**), birds or fish (claimed). Such compositions can be used to prevent or treat bacterial, viral, mycoplasmal, mycobacterial, yeast or parasitic infections, especially systemic infections or infections at a mucosal surface (claimed). They are especially useful to prevent or treat **human** infection with **human** immunodeficiency virus (HIV), **respiratory syncytial virus**, flu virus or cold virus (claimed). SIgA is usually found in external secretions such as colostrum, saliva, tears etc. and is often the first line of defence against infectious agents in the body.

ADVANTAGE - The method allows production of commercial quantities of sIg molecules for therapeutic use, not previously possible; production using non-plant cells and a single cell type is more efficient than a previous multi-step process of fusing **recombinant** plant cells, and avoids alterations of the sIg by plant cells. SIgA molecules are more stable and resistant to proteolysis than previously used IgA molecules, and can be administered to prevent as well as to treat infections, unlike e.g. IgG and IgM molecules.
Dwg.0/4

L24 ANSWER 58 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 1998-110579 [10] WPIDS

DOC. NO. CPI: C1998-036424
TITLE: Attenuated **respiratory syncytial virus** vaccines - useful to protect individuals against **RSV** infection.
DERWENT CLASS: B04 D16
INVENTOR(S): BUKREYEV, A A; COLLINS, P L; JUHASZ, K; MURPHY, B R; TENG, M N; WHITEHEAD, S S; COLLINS, P I
PATENT ASSIGNEE(S): (USSH) US DEPT HEALTH & HUMAN SERVICES
COUNTRY COUNT: 79
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9802530	A1	19980122	(199810)*	EN	242
RW: AT BE CH DE DK EA ES FI FR GB GH GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW					
AU 9737997	A	19980209	(199823)		
EP 912724	A1	19990506	(199922)	EN	
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU NL PT SE					
CN 1224462	A	19990728	(199948)		
US 5993824	A	19991130	(200003)		
BR 9710363	A	20000111	(200020)		
KR 2000023802	A	20000425	(200107)		
AU 2001055916	A	20010920	(200166)#		
AU 2001055922	A	20010920	(200166)#		
JP 2002511731	W	20020416	(200242)		240

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 9802530	A1		WO 1997-US12269	19970715
AU 9737997	A		AU 1997-37997	19970715
EP 912724	A1		EP 1997-934948	19970715
			WO 1997-US12269	19970715
CN 1224462	A		CN 1997-196139	19970715
US 5993824	A	Provisional	US 1996-21773P	19960715
		Provisional	US 1997-46141P	19970509
		Provisional	US 1997-47634P	19970523
			US 1997-892403	19970715
BR 9710363	A		BR 1997-10363	19970715
			WO 1997-US12269	19970715
KR 2000023802	A		KR 1999-700286	19990115
AU 2001055916	A	Div ex	AU 1997-37997	19970715
			AU 2001-55916	20010723
AU 2001055922	A	Div ex	AU 1997-37997	19970715
			AU 2001-55922	20010723
JP 2002511731	W		WO 1997-US12269	19970715
			JP 1998-506235	19970715

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9737997	A	Based on	WO 9802530
EP 912724	A1	Based on	WO 9802530

BR 9710363 A Based on WO 9802530
JP 2002511731 W Based on WO 9802530

PRIORITY APPLN. INFO: US 1997-47634P 19970523; US 1996-21773P 19960715; US 1997-46141P 19970509; US 1997-892403 19970715; AU 2001-55916 20010723; AU 2001-55922 20010723

AN 1998-110579 [10] WPIDS
AB WO 9802530 A UPAB: 19980309
A new isolated infectious **recombinant respiratory syncytial virus** (**RSV**) comprises a **RSV** genome or antigenome, a major nucleocapsid (N) protein, a nucleocapsid phosphoprotein (P), a large polymerase protein (L), and a RNA polymerase elongation factor, where the **recombinant RSV** has at least two attenuating mutations, one of the mutations specifying a temperature-sensitive (ts) substitution at amino acid Phe521, Gln831, Met1169 or Tyr1321 in the **RSV** polymerase gene or a ts nucleotide substitution in the gene-start sequence of gene M2.

Also claimed are: (1) an isolated infectious **RSV** particle which comprises a **recombinant RSV** (anti)genome, N, P, and L proteins, a RNA polymerase elongation factor, where the (anti)genome is modified: (i) to ablate or modulate expression of a SH, NS1, NS2 or G gene or a cis-acting regulatory sequence; and (ii) by a termination codon introduced within a selected gene, or by a change in sequence, position or presence of a GS or GE transcription signal relative to the selected gene; (2) a composition which comprises an expression vector comprising an isolated **polynucleotide** encoding a **RSV** genome or antigenome having at least two attenuating mutations as above, and one or more expression vectors which comprises one or more **polynucleotide** molecules encoding N, P, L and RNA polymerase elongation factor proteins of **RSV**, where upon expression an infectious **RSV** particle is produced; and (3) an **RSV** strain selected from cpts **RSV** 248 (ATCC VR 2450), cpts 248/404 (ATCC VR 2454), cpts 248/955 (ATCC VR 2453), cpts **RSV** 530 (ATCC VR 2452), cpts 530/1009 (ATCC VR 2451) or cpts 530/1030 (ATCC VR 2455), or B-1 cp52/2B5 (ATCC VR 2542) or B-1 cp-23 (ATCC VR).

USE - The isolated attenuated **recombinant RSV** and **RSV** particles are used (in a vaccine) to stimulate the immune system of an individual to induce protection against **respiratory syncytial virus**. In particular the attenuated **RSV** is administered to an individual seronegative for antibodies to **RSV** or possessing trans-placentally acquired maternal antibodies to **RSV**. The expression vector of (2) is used for the production of infectious attenuated **RSV** particles. (All claimed).

The attenuated **RSV** is formulated in a dose of 103 to 106 and administered to the upper respiratory tract by spray, droplet or aerosol (claimed).

ADVANTAGE - The immune system of an individual can be stimulated to induce protection against natural **RSV** infection, or multivalently against infection by **RSV** and another pathogen, such as **parainfluenza** virus (PIV) by administration of attenuated, biologically derived or **recombinant RSV**.
Dwg.0/23

L24 ANSWER 59 OF 67 MEDLINE DUPLICATE 8
ACCESSION NUMBER: 1998451578 MEDLINE
DOCUMENT NUMBER: 98451578 PubMed ID: 9776746
TITLE: Unstable retrovirus **mutants** with acquired transforming activity: rapid changes in the number of

repeats of a specific junD **polynucleotide** segment.
AUTHOR: Ito T; Kabuyama Y; Okazaki S; Kameda T; Murakami M; Iba H
CORPORATE SOURCE: Department of Gene Regulation, Institute of Medical Science, University of Tokyo, 4-6-1 Shirokanedai, Minato-ku, Tokyo 108-8639, Japan.
SOURCE: NUCLEIC ACIDS RESEARCH, (1998 Nov 1) 26 (21) 4868-73. Journal code: 0411011. ISSN: 0305-1048.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199812
ENTRY DATE: Entered STN: 19990115
Last Updated on STN: 19990115
Entered Medline: 19981223

AB We have previously reported that the non-transforming jun D (wild type) gene can acquire transforming activity through spontaneous mutations when it is replicated through avian replication-competent retrovirus vectors in chicken embryo fibroblasts. In two of these spontaneous **mutants**, T1 and T2, which were isolated from proviral DNA in the same transformed cell clone, a specific 48 bp **polynucleotide** segment of the jun D coding sequence was tandemly repeated three and five times, respectively. We report here that the number of direct repeats in these **mutants** rapidly changes (mostly decreases) in the context of either **RSV**-based replication-competent or MLV-based replication-defective retroviruses, most likely during the process of reverse transcription, while these mutations are stable in the cellular chromosome. We also show that the growth conditions of the infected culture modulate the proportions of polymorphic proviral populations in the infected culture. We finally discuss the possible molecular mechanisms that generate genetic diversity in these amplification **mutants**.

L24 ANSWER 60 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 1997-549730 [50] WPIDS
DOC. NO. NON-CPI: N1997-458329
DOC. NO. CPI: C1997-175369
TITLE: Purified GC-binding factor 2 protein - used to inhibit expression of particularly the epidermal growth factor receptor gene, especially for treating cancer.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): JOHNSON, A C
PATENT ASSIGNEE(S): (USSH) US DEPT HEALTH & HUMAN SERVICES
COUNTRY COUNT: 76
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9741226	A2	19971106	(199750)*	EN	74
RW: AT BE CH DE DK EA ES FI FR GB GH GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TR TT UA UG US UZ VN YU					
AU 9729935	A	19971119	(199812)		
WO 9741226	A3	19971231	(199817)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE

WO 9741226	A2	WO 1997-US7172	19970428
AU 9729935	A	AU 1997-29935	19970428
WO 9741226	A3	WO 1997-US7172	19970428

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9729935	A	Based on	WO 9741226

PRIORITY APPLN. INFO: US 1996-16465P 19960429
AN 1997-549730 [50] WPIDS
AB WO 9741226 A UPAB: 19971217
A purified GCF2 (GC-binding factor 2) protein GCF2 having a sequence (practically) identical to the 752 amino acid (aa) sequence given in the specification, is new. Also claimed are: (1) analogues (Ia) of GCF2 with a non-natural sequence but including at least 10 contiguous aa from the 752 aa sequence; (2) a **recombinant** nucleic acid (II) which: (i) contains at least 25 contiguous nucleotides (nt) from regions 128-1384 or 1694-2310 of a 3523 bp cDNA sequence of GCF2 given in the specification; or (ii) encodes a polypeptide (Ib) containing at least 10 contiguous aa of the 752 aa sequence; (3) a host cell transformed with a vector expressing (Ib); (4) a **polynucleotide** probe containing at least 15 nt which are able to hybridise specifically with the 3523 bp sequence or its complement; (5) an antibody (Ab) that binds native GCF2 specifically; and (6) a method for detecting GCF2 by formation of a complex with a binding substrate (A).

USE - GCF2, (Ia) and (Ib) are used to raise the Ab of (5). They also inhibit expression in a cell of nucleic acids linked to a EGFR (epidermal growth factor receptor), **RSV** (Rous sarcoma virus) or SV40 promoter, particularly by transfection of the cell with nucleic acid expressing (Ib). Especially this method is used to inhibit expression of EGFR, preferably in cancer cells that overexpress it, also to restore GCF2-binding activity to cancer cells deficient in this activity. GCF2 can also be used to isolate DNA sequences that bind to it from DNA libraries. The cells of (3) are used for **recombinant** expression of GCF2 or (Ia). The probes of (4) are used to detect GCF2-related cDNA and mRNA (either by reverse-transcription PCR or by in situ hybridisation with a labelled probe). Detection of this mRNA may be used to diagnose, e.g. cancer (especially of the breast, B cell lymphoma or T cell lymphoma), detected from elevated levels of a 4.2 kb transcript but depressed levels of a 2.4 kb transcript. Labelled probes can be used to detect chromosomal translocations of the GCF2 gene and polymorphic forms of the gene are detected by comparing nt or aa at specific positions with those in the wild type (all claimed). The Ab are useful as immunoassay reagents, for purifying GCF2 and for raising anti-idiotypic antibodies.
Dwg.0/20

L24 ANSWER 61 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 1997-212893 [19] WPIDS
DOC. NO. CPI: C1997-068811
TITLE: Infectious **respiratory syncytial virus** particles - useful for treatment of **RSV** or gene therapy of upper respiratory tract diseases.
DERWENT CLASS: B04 D16
INVENTOR(S): COLLINS, P L
PATENT ASSIGNEE(S): (USSH) US DEPT HEALTH & HUMAN SERVICES; (COLL-I) COLLINS P L

COUNTRY COUNT: 75
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9712032	A1	19970403	(199719)*	EN	66
RW: AT BE CH DE DK EA ES FI FR GB GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE HU IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK TJ TM TR TT UA UG US UZ VN					
AU 9671192	A	19970417	(199732)		
EP 859831	A1	19980826	(199838)	EN	
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					
JP 11512609	W	19991102	(200003)		64
KR 99063811	A	19990726	(200043)		
AU 727923	B	20010104	(200107)		
US 6264957	B1	20010724	(200146)		
US 2002182228	A1	20021205	(200301)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 9712032	A1		WO 1996-US15524	19960927
AU 9671192	A		AU 1996-71192	19960927
EP 859831	A1		EP 1996-932349	19960927
			WO 1996-US15524	19960927
JP 11512609	W		WO 1996-US15524	19960927
			JP 1997-513678	19960927
KR 99063811	A		WO 1996-US15524	19960927
			KR 1998-702279	19980327
AU 727923	B		AU 1996-71192	19960927
US 6264957	B1	Provisional	US 1995-7083P	19950927
			US 1996-720132	19960927
US 2002182228	A1	Provisional	US 1995-7083P	19950927
		Div ex	US 1996-720132	19960927
			US 2001-847173	20010503

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9671192	A	Based on	WO 9712032
EP 859831	A1	Based on	WO 9712032
JP 11512609	W	Based on	WO 9712032
KR 99063811	A	Based on	WO 9712032
AU 727923	B	Previous Publ.	AU 9671192
		Based on	WO 9712032
US 2002182228	A1	Div ex	US 6264957

PRIORITY APPLN. INFO: US 1995-7083P 19950927; US 1996-720132 19960927; US 2001-847173 20010503
AN 1997-212893 [19] WPIDS
AB WO 9712032 A UPAB: 19970512
An isolated infectious **respiratory syncytial virus** (**RSV**) particle comprises a **recombinant RSV** genome or anti-genome, a nucleocapsid protein (N), a nucleocapsid phosphoprotein (P), a large polymerase protein (L) and an RNA elongation factor (E).

Also claimed are: (1) an isolated **polynucleotide** (PN) molecule which comprises an operably linked transcriptional promoter, a PN encoding an **RSV** (anti)genome, and a transcriptional terminator, and (2) a cell or cell-free extract contg. (i) an expression vector comprising a PN encoding an **RSV** (anti)genome; and (ii) an expression vector comprising a PN encoding the N, P, L or E proteins, where expression of the **RSV** genome and the N, P, L and E proteins combine to produce an infectious **RSV** particle.

USE - **Human RSV** is an important paediatric pathogen world-wide. Generation of **RSV** particles containing a minimum of sequence which allow it to be infective can be useful for generating vaccines against **RSV**. The ability to introduce defined mutations in the **RSV** genome can be used to study the virus molecular biology and pathology. The viruses can also be used for gene therapy of the upper respiratory tract.

Dwg.0/2

L24 ANSWER 62 OF 67 MEDLINE DUPLICATE 9

ACCESSION NUMBER: 1998054343 MEDLINE
DOCUMENT NUMBER: 98054343 PubMed ID: 9391135
TITLE: **Respiratory syncytial virus** (**RSV**) SH and G proteins are not essential for viral replication in vitro: clinical evaluation and molecular characterization of a cold-passaged, attenuated **RSV** subgroup B **mutant**.
AUTHOR: Karron R A; Buonagurio D A; Georgiu A F; Whitehead S S; Adamus J E; Clements-Mann M L; Harris D O; Randolph V B; Udem S A; Murphy B R; Sidhu M S
CORPORATE SOURCE: Center for Immunization Research, Department of International Health, School of Hygiene and Public Health, The Johns Hopkins University, Baltimore, MD 21205, USA.. rkarron@jhsph.edu
CONTRACT NUMBER: AI-15095 (NIAID)
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1997 Dec 9) 94 (25) 13961-6. Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199801
ENTRY DATE: Entered STN: 19980129
Last Updated on STN: 19980129
Entered Medline: 19980115

AB A live, cold-passaged (cp) candidate vaccine virus, designated **respiratory syncytial virus** (**RSV**) B1 cp-52/2B5 (cp-52), replicated efficiently in Vero cells, but was found to be overattenuated for **RSV**-seronegative infants and children. Sequence analysis of reverse-transcription-PCR-amplified fragments of this **mutant** revealed a large deletion spanning most of the coding sequences for the small hydrophobic (SH) and attachment (G) proteins. Northern blot analysis of cp-52 detected multiple unique read-through mRNAs containing SH and G sequences, consistent with a **deletion** mutation spanning the **SH**:G gene junction. Immunological studies confirmed that an intact G glycoprotein was not produced by the cp-52 virus. Nonetheless, cp-52 was infectious and replicated to high titer in tissue culture despite the absence of the viral surface SH and G glycoproteins. Thus, our characterization of this negative-strand RNA virus identified a novel replication-competent deletion **mutant** lacking two of its three surface glycoproteins. The requirement of SH and

G for efficient replication in vivo suggests that selective deletion of one or both of these **RSV** genes may provide an alternative or additive strategy for developing an optimally attenuated vaccine candidate.

L24 ANSWER 63 OF 67 MEDLINE DUPLICATE 10
ACCESSION NUMBER: 1998037604 MEDLINE
DOCUMENT NUMBER: 98037604 PubMed ID: 9371553
TITLE: **Recombinant respiratory syncytial virus** from which the entire **SH** gene has been **deleted** grows efficiently in cell culture and exhibits site-specific attenuation in the respiratory tract of the mouse.
AUTHOR: Bukreyev A; Whitehead S S; Murphy B R; Collins P L
CORPORATE SOURCE: Laboratory of Infectious Diseases, National Institute of Allergy and Infectious Diseases, Bethesda, Maryland 20892-0720, USA.
SOURCE: JOURNAL OF VIROLOGY, (1997 Dec) 71 (12) 8973-82. Journal code: 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199712
ENTRY DATE: Entered STN: 19980116
Last Updated on STN: 19980116
Entered Medline: 19971224

AB The small hydrophobic protein SH of **human respiratory syncytial virus** (**RSV**) is a short transmembrane surface protein of unknown function. A full-length cDNA of **RSV** strain A2 (subgroup A) antigenomic RNA was modified such that the entire SH gene, including the transcription signals and the complete mRNA-encoding sequence, was deleted and replaced by a synthetic intergenic region. This reduced the length of the antigenome by 398 nucleotides and ablated expression of 1 of the 10 **RSV** mRNAs. **Recombinant** virus containing this engineered deletion was recovered, and the absence of the SH gene was confirmed by reverse transcription in conjunction with PCR. Northern blot analysis of intracellular RNAs and gel electrophoresis of labeled intracellular proteins confirmed the lack of expression of the SH mRNA and protein. The absence of the SH gene did not noticeably affect RNA replication, but two effects on transcription were noted. First, synthesis of the G, F, and M2 mRNAs was increased, presumably due to their being one position closer to the promoter in the gene order. Second, transcription of genes downstream of the engineered site exhibited a steeper gradient of polarity. On monolayers of HEp-2 cells, the SH-minus virus produced syncytia which were at least equivalent in size to those of the wild type and produced plaques which were 70% larger. Furthermore, the SH-minus virus grew somewhat better (up to 12.6-fold) than wild-type **recombinant RSV** in certain cell lines. While the function of the SH protein remains to be determined, it seems to be completely dispensable for growth in tissue culture and fusion function. When inoculated intranasally into mice, the SH-minus virus resembled the wild-type **recombinant** virus in its efficiency of replication in the lungs, whereas it replicated 10-fold less efficiently in the upper respiratory tract. In mice, the SH-minus and wild-type **recombinant** viruses were similarly immunogenic and effective in inducing resistance to virus challenge.

L24 ANSWER 64 OF 67 WPIDS (C) 2003 THOMSON DERWENT
ACCESSION NUMBER: 1995-022311 [03] WPIDS

DOC. NO. NON-CPI: N1995-017433
DOC. NO. CPI: C1995-010272
TITLE: Eliciting immune responses in an animal - using a complex of a cationic lipid with a **polynucleotide** coding for an antigenic determinant.
DERWENT CLASS: B04 C06 D16 S03
INVENTOR(S): HEARL, W G; JESSEE, J A
PATENT ASSIGNEE(S): (LIFE-N) LIFE TECHNOLOGIES INC; (HEAR-I) HEARL W G; (JESS-I) JESSEE J A
COUNTRY COUNT: 19
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9427435	A1	19941208	(199503)*	EN	39
RW: AT BE CH DE DK ES FR GB GR IE IT LU MC NL PT SE					
W: JP					
EP 702516	A1	19960327	(199617)	EN	
R: AT BE CH DE DK ES FR GB GR IE IT LI LU MC NL PT SE					
JP 09500013	W	19970107	(199711)		36
US 2002077305	A1	20020620	(200244)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 9427435	A1		WO 1994-US6105	19940531
EP 702516	A1		EP 1994-918673	19940531
			WO 1994-US6105	19940531
JP 09500013	W		WO 1994-US6105	19940531
			JP 1995-501040	19940531
US 2002077305	A1	Cont of	US 1993-69720	19930601
			US 1995-450555	19950525

FILING DETAILS:

PATENT NO	KIND		PATENT NO
EP 702516	A1	Based on	WO 9427435
JP 09500013	W	Based on	WO 9427435

PRIORITY APPLN. INFO: US 1993-69720 19930601; US 1995-450555 19950525
AN 1995-022311 [03] WPIDS
AB WO 9427435 A UPAB: 19950126
The following are claimed: (A) eliciting an immune response in an animal comprising: (a) mixing 1 cationic lipid (CL) with a **polynucleotide** (PN) coding for an antigenic determinant, thereby forming a CL-PN complex and (b) administering the complex to the animal; (B) producing polyclonal antibodies (PAbs) to an immunogen in an animal, comprising: (a) mixing 1 CL with a PN coding for the immunogen, thereby forming a CL-PN complex, (b) administering the complex to the animal and (c) isolating the PAbs from the animal; (C) producing monoclonal antibodies (MAbs) comprising: (a) step (a) as in (B), (b) administering the complex to 1 mouse, (c) removing B-lymphocytes from the immunised mice, (d) fusing the B-lymphocytes with myeloma cells, thereby producing hybridomas, (e) cloning the hybridomas, (f) selecting positive clones which produce anti-immunogen antibody, (g) culturing the anti-immunogen antibody-producing clones and (h) isolating anti-immunogen antibodies from the cultures; (D) mapping the epitopes of a protein molecule, comprising:

(a) fragmenting DNA molecules coding for the protein molecule in a random manner, (b) subcloning the DNA fragments in an expression vector, (c) mixing 1 CL with each of the expression vector subclones, thereby forming a CL-expression vector complex with each of the expression vector subclones, (d) administering the complexes to mice and (e) determining which of the DNA fragments are capable of generating the prodn. of antibodies in the mice; (E) generating active immunity against an infectious disease in an animal, comprising: (a) mixing 1 CL with a PN coding for an antigenic determinant of an organism which is the causative agent of the infectious disease, thereby forming a CL-PN complex and (b) administering the complex to the animal, whereby active immunity to the infectious disease is generated.

PREFERRED CATIONIC LIPID - The CL may be e.g. Lipofectamine, Lipofectace, 1-propanaminium, N-(2-(2-bromo)ethyl)-N,N-dimethyl-2,3-bis(9-octadecenyloxy)bromide, DORI-ether or DORI-ether lysolipid. PREFERRED VECTOR - The **polynucleotide** is either (i) an RNA molecule coding for an immunogen, (ii) a **recombinant** DNA/RNA mol. with a promoter (e.g. SV40, **RSV**, CMV) and an immunogen coding sequence.

ADVANTAGE - The CL-PN complexes provide an efficient method for genetic immunisation. The use of CLs as carriers for DNA constructs permit genetic immunisation with as little as 5mug of a DNA construct. Dwg.0/0

L24 ANSWER 65 OF 67 MEDLINE DUPLICATE 11
ACCESSION NUMBER: 87165921 MEDLINE
DOCUMENT NUMBER: 87165921 PubMed ID: 2435721
TITLE: Interactions of retroviral structural proteins with single-stranded nucleic acids.
AUTHOR: Karpel R L; Henderson L E; Oroszlan S
CONTRACT NUMBER: GM-32370 (NIGMS)
N01-CO-23909 (NCI)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1987 Apr 15) 262 (11) 4961-7.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198705
ENTRY DATE: Entered STN: 19900303
Last Updated on STN: 19970203
Entered Medline: 19870515

AB We have studied the interactions of single-stranded polyribonucleotides with murine leukemia virus structural proteins p10, p10' (a p10 **variant**), and Pr65gag, as well as Rous sarcoma virus (**RSV**) pp12 (a p10 analog). Two quantitative assays have been used to monitor protein-RNA association: the fluorescence enhancement of polyethenoadenylic acid) poly(epsilon A) upon binding protein, and tryptophan fluorescence quenching upon binding to poly(U). With each assay p10 was shown to bind stoichiometrically to single-stranded RNA, covering a length of nucleic acid chain (occluded site size, n) of about 6 residues. **RSV** pp12 was also shown to bind to poly(epsilon A), with n = 5 +/- 1. Addition of NaCl to fully titrated MuLV p10-nucleic acid mixtures effected nearly complete restoration of poly(epsilon A) or MuLV p10 fluorescence. Under conditions of 0.06 M NaCl, p10 bound noncooperatively to poly(epsilon A) with an intrinsic association constant, K = 2.3 X 10(6) M-1. K and n determined in this study were shown to relate to Kapp determined by other methods, by the approximation Kapp approximately NK, where N is the number of binding sites along the

polynucleotide chain ((nucleotides/chain)/n). Chemical modifications of the p10 cysteine residues did not alter the affinity for poly(epsilon A). The affinity of Pr65gag for poly(epsilon A) appears to be higher than that of p10.

L24 ANSWER 66 OF 67 MEDLINE
ACCESSION NUMBER: 79033816 MEDLINE
DOCUMENT NUMBER: 79033816 PubMed ID: 212741
TITLE: Transfer of duck cell DNA sequences to the nucleus of 3T3 cells by Rous sarcoma virus.
AUTHOR: Baxt W G; Meinkoth J L
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1978 Sep) 75 (9) 4252-6. Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197812
ENTRY DATE: Entered STN: 19900314
Last Updated on STN: 19970203
Entered Medline: 19781227

AB Peking duck cell nuclear DNA has no complementarity to RNA of Prague C Rous sarcoma virus (**RSV**). Upon infection of Peking duck cells by Prague C **RSV**, **polynucleotide** sequences complementary to Peking duck cell nuclear DNA can be detected in the high molecular weight RNA from progeny Peking duck cell passaged **RSV** (RSVPD) by RNA.DNA molecular hybridazation. When 3T3 cells are subsequently infected by RSVPD, **polynucleotide** sequences complementary to Peking duck cell nuclear DNA can be detected in the 3T3 cell nuclear DNA by RNA.DNA molecular hybridization. The potential consequences of the transfer of the Peking duck cell nuclear DNA from the avian to the murine cells are discussed.

L24 ANSWER 67 OF 67 MEDLINE
ACCESSION NUMBER: 69087302 MEDLINE
DOCUMENT NUMBER: 69087302 PubMed ID: 5762783
TITLE: Influenza virus: genetics and control.
AUTHOR: Simpson R W
SOURCE: SCIENCE, (1969 Jan 24) 163 (865) 409-12. Journal code: 0404511. ISSN: 0036-8075.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 196902
ENTRY DATE: Entered STN: 19900101
Last Updated on STN: 19970203
Entered Medline: 19690228

STIC Search

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=> d his full

     FILE 'HCAPLUS' ENTERED AT 16:00:32 ON 05 MAY 2003
L15         4045 SEA ABB=ON  (?RESPIRATORY?(W)?SYNCYTIAL?(W)?VIRUS? OR RSV)
L16         1148 SEA ABB=ON  L15 AND (?RECOMB? OR ?VARIANT? OR ?MUTANT?)
L17           11 SEA ABB=ON  L16 AND (?DELET?(5A)(?SMALL?(W)?HYDROPHOB? OR SH))

                 D AU 1-11
L18           16 SEA ABB=ON  L16 AND ?POLYNUCLEOTID?
                 D AU 1-16
L19           26 SEA ABB=ON  L17 OR L18
                 D TI 1-26
L20           19 SEA ABB=ON  L19 AND ?HUMAN?
L21            7 SEA ABB=ON  L19 AND ?PARAINFLUENZ?
L22           26 SEA ABB=ON  L19 OR L20 OR L21

     FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, JICST-EPLUS, JAPIO' ENTERED AT
     16:08:12 ON 05 MAY 2003
L23           87 SEA ABB=ON  L22
L24           67 DUP REMOV L23 (20 DUPLICATES REMOVED)

     FILE 'HCAPLUS' ENTERED AT 16:12:57 ON 05 MAY 2003

L25            1 SEA ABB=ON  L16 AND ?POLYNUCLEOTID?(5A)(?RESPIRATORY?(W)?SYNCYT
                 IAL?(W)?VIRUS? OR RSV)

     FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, JICST-EPLUS, JAPIO' ENTERED AT
     16:15:46 ON 05 MAY 2003
L26           11 SEA ABB=ON  L25
L27           11 DUP REMOV L26 (0 DUPLICATES REMOVED)
L28           67 SEA ABB=ON  L24 OR L27 67 cits from "other databases"

     FILE 'HCAPLUS' ENTERED AT 16:18:36 ON 05 MAY 2003
L29           26 SEA ABB=ON  L22 OR L25 26 cits from CA Plus—
                                        These appear first in attached
                                        printout
```

Inventor Search

=> d ibib abs ind 1-2

L14 ANSWER 1 OF 2 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2000:72434 HCAPLUS
DOCUMENT NUMBER: 133:41730
TITLE: Rational design of live-attenuated recombinant vaccine virus for human respiratory syncytial virus by reverse genetics
AUTHOR(S): **Collins, Peter L.; Whitehead, Stephen S.; Bukreyev, Alexander;** Fearns, Rachel; **Teng, Michael N.; Juhasz, Katalin;** Chanock, Robert M.; **Murphy, Brian R.**
CORPORATE SOURCE: Laboratory of Infectious Diseases, National Institute of Allergy and Infectious Diseases, Bethesda, MD, 20892-0720, USA
SOURCE: Advances in Virus Research (1999), 54, 423-451
CODEN: AVREA8; ISSN: 0065-3527
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with 49 refs. Discussed are: respiratory syncytial virus (RSV); RSV disease; immunity to RSV; obstacles to development of RSV vaccine; RSV reverse genetics (antigenic subgroup A); expression of foreign genes in recombinant RSV; gene knockouts; biol. derived RSV subgroup A candidate vaccine viruses; rational design of recombinant subgroup A vaccine; advantages of recombinant DNA method; and current directions. (c) 1999 Academic Press.
CC 15-0 (Immunochemistry)
ST vaccine respiratory syncytial virus reverse genetics review
IT Genetics
(mol., reverse; rational design of live-attenuated recombinant vaccine virus for human respiratory syncytial virus by reverse genetics)
IT Respiratory syncytial virus
Vaccines
(rational design of live-attenuated recombinant vaccine virus for human respiratory syncytial virus by reverse genetics)
REFERENCE COUNT: 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 2 OF 2 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1998:71212 HCAPLUS
DOCUMENT NUMBER: 128:151668
TITLE: Attenuated respiratory syncytial virus with a temperature-sensitive mutation in the polymerase and their use in vaccines
INVENTOR(S): **Murphy, Brian R.; Collins, Peter L.; Whitehead, Stephen S.; Bukreyev, Alexander A.; Juhasz, Katalin; Teng, Michael N.**
PATENT ASSIGNEE(S): United States Dept. of Health and Human Services, USA; Murphy, Brian R.; Collins, Peter L.; Whitehead, Stephen S.; Bukreyev, Alexander A.; Juhasz, Katalin; Teng, Michael N.
SOURCE: PCT Int. Appl., 242 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9802530	A1	19980122	WO 1997-US12269	19970715
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
CA 2257823	AA	19980122	CA 1997-2257823	19970715
AU 9737997	A1	19980209	AU 1997-37997	19970715
EP 912724	A1	19990506	EP 1997-934948	19970715
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, PT, IE, FI				
CN 1224462	A	19990728	CN 1997-196139	19970715
US 5993824	A	19991130	US 1997-892403	19970715
BR 9710363	A	20000111	BR 1997-10363	19970715
JP 2002511731	T2	20020416	JP 1998-506235	19970715
KR 2000023802	A	20000425	KR 1999-700286	19990115
PRIORITY APPLN. INFO.:			US 1996-21773P P	19960715
			US 1997-46141P P	19970509
			US 1997-47634P P	19970523
			WO 1997-US12269 W	19970715

AB Attenuated respiratory syncytial virus suitable for use in vaccines are prepd. by introducing mutations assocd. with attenuated phenotypes into a host virus. The virus may also carry genes for protective antigens of related viruses, such as parainfluenza virus. The host may be a wild type or a strain that has been incompletely attenuated by cold passage. The mutations may have temp.-sensitive (ts) or cold-adapted (ca) phenotypes. Attenuation is greatest when one of the mutations is a ts mutant of the viral polymerase gene. Mutation in the M2 gene also leads to attenuation. The mutations can be naturally-occurring, chem.-induced, or introduced by site-specific mutagenesis. Characterizations of a no. of vaccine strains for stability and effectiveness in animal hosts are reported.

IC ICM C12N007-04
ICS C12N007-01; A61K039-155; C12N015-45; C12N007-00

CC 10-4 (Microbial, Algal, and Fungal Biochemistry)
Section cross-reference(s): 3, 15

ST attenuated respiratory syncytial virus vaccine

IT Proteins, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(F, parainfluenza virus gene for, in attenuated respiratory syncytial virus vaccine strain; attenuated respiratory syncytial virus with temp.-sensitive mutation in polymerase and their use in vaccines)

IT Gene, microbial
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(G, attenuation of respiratory syncytial virus by mutation in; attenuated respiratory syncytial virus with temp.-sensitive mutation in polymerase and their use in vaccines)

IT Glycoproteins, specific or class
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(HN (hemagglutinin-neuraminidase), parainfluenza virus gene for, in attenuated respiratory syncytial virus vaccine strain; attenuated respiratory syncytial virus with temp.-sensitive mutation in polymerase and their use in vaccines)

IT Proteins, specific or class
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(L, attenuating mutants in genes for; attenuated respiratory syncytial virus with temp.-sensitive mutation in polymerase and their use in vaccines)
IT Transcription factors
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(M2, attenuating mutants in genes for; attenuated respiratory syncytial virus with temp.-sensitive mutation in polymerase and their use in vaccines)
IT Proteins, specific or class
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(N (nucleocapsid); attenuated respiratory syncytial virus with temp.-sensitive mutation in polymerase and their use in vaccines)
IT Gene, microbial
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(NS1, attenuation of respiratory syncytial virus by mutation in; attenuated respiratory syncytial virus with temp.-sensitive mutation in polymerase and their use in vaccines)
IT Gene, microbial
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(NS2, attenuation of respiratory syncytial virus by mutation in; attenuated respiratory syncytial virus with temp.-sensitive mutation in polymerase and their use in vaccines)
IT Gene, microbial
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(SH, attenuation of respiratory syncytial virus by mutation in; attenuated respiratory syncytial virus with temp.-sensitive mutation in polymerase and their use in vaccines)
IT Epitopes
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(T-helper, gene for, in attenuated respiratory syncytial virus vaccine strain; attenuated respiratory syncytial virus with temp.-sensitive mutation in polymerase and their use in vaccines)
IT Drug delivery systems
(aerosols, for delivery of vaccine strains of respiratory syncytial virus; attenuated respiratory syncytial virus with temp.-sensitive mutation in polymerase and their use in vaccines)
IT Human respiratory syncytial virus
(attenuated respiratory syncytial virus with temp.-sensitive mutation in polymerase and their use in vaccines)
IT Genetic element
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(cis regulatory element, attenuation of respiratory syncytial virus by mutation in; attenuated respiratory syncytial virus with temp.-sensitive mutation in polymerase and their use in vaccines)
IT Mutation
(cold-adapted, in attenuated respiratory syncytial virus; attenuated respiratory syncytial virus with temp.-sensitive mutation in polymerase and their use in vaccines)
IT Cytokines
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(gene for, in attenuated respiratory syncytial virus vaccine strain; attenuated respiratory syncytial virus with temp.-sensitive mutation in polymerase and their use in vaccines)
IT RNA sequences

(of human respiratory syncytial virus; attenuated respiratory syncytial virus with temp.-sensitive mutation in polymerase and their use in vaccines)

IT Vaccines
(respiratory syncytial virus; attenuated respiratory syncytial virus with temp.-sensitive mutation in polymerase and their use in vaccines)

IT Mutation
(temp.-sensitive, in attenuated respiratory syncytial virus; attenuated respiratory syncytial virus with temp.-sensitive mutation in polymerase and their use in vaccines)

IT Human parainfluenza virus
Human parainfluenza virus 1
Human parainfluenza virus 2
Human parainfluenza virus 3
(vaccines against; attenuated respiratory syncytial virus with temp.-sensitive mutation in polymerase and their use in vaccines)

IT 9026-28-2, RNA-dependent RNA polymerase
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(attenuating mutants in genes for; attenuated respiratory syncytial virus with temp.-sensitive mutation in polymerase and their use in vaccines)

IT 202606-70-0
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(nucleotide sequence; attenuated respiratory syncytial virus with temp.-sensitive mutation in polymerase and their use in vaccines)

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT